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A CASE OF REFLEX SYMPATHETIC DYSTROPHY RELIEVED BY SYMPATHECTOMY*

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NEW ORLEANS

This case is presented because it exhibits an interesting diagnostic problem and because it belongs, I believe, to a group of conditions which are little understood, and therefore, frequently mishandled.

CASE REPORT

W. J. M., a thirty-eight-year-old white male, a cable splicer by occupation, was admitted to the Veterans Hospital at New Orleans on December 18, 1947. His chief complaint was burning pain in the right shoulder, arm, and forearm. Symptoms had begun in March, 1944, when he had noted a sharp, stinging sensation in the right forearm while loading boxes on a truck. This had been associated with a soft swelling about the size of a lemon on the posterolateral aspect of the forearm in its upper third. Shortly afterwards, other tender nodules had appeared over posterior and lateral aspects of forearm, the lateral aspect of the arm, and the right scapular area. These nodules had subsided after a few days, leaving a small, firm, slightly tender subcutaneous lump. About one month after onset, he had begun to have pain over these areas. The pain was described as stinging and burning as if ants were crawling under the skin. It was continuous and aggravated by exercise. Eight weeks after onset, he had noted that the skin over the painful areas was very elastic, that the subcutaneous fat was lacking, and the involved area was very sensitive to touch. Symptoms had persisted after discharge from the Army in July, 1944, but during the past few months had become more severe, worse at night, and incapacitating. Since February, 1945, he had noted no increase in the area involved. There was no loss of strength in the right upper extremity, but after exertion, it tired easily and the hand became swollen. The patient had no symptoms other than those related in the present illness.

On physical examination temperature, pulse, and respiration were normal and the blood pressure was found to be 150/100 in the right arm. The pressure in the left arm was approximately the same, 148/96.

The patient was a well developed, well nourished white male who did not appear acutely ill. He was well orientated and seemed to be sincere and emotionally stable. The parts involved were the right scapular region, lateral half of right arm, posterolateral aspect of right forearm, the lateral half of dorsum of the hand and the right index and ring fingers. Over these areas the skin was paper thin and very elastic. Subcutaneous fat was lacking and sensitivity to touch marked. Over the arm, forearm, and hand were numerous dilated, tortuous veins. In the scapular region were multiple depressed, pigmented points. Pinkish-brown discoloration and definite increased temperature were present over arm and forearm. Muscular power and reflexes were normal. The brachial and radial pulsations were equal and of normal volume on the two sides. No bruit or thrill could be found. Adson's test was negative.

Blood and urine examinations were normal. The Kahn was negative.

X-ray studies of the chest, cervical spine, shoulder girdle, and upper extremities revealed no abnormalities except two very small cystic areas noted in the head of the first metacarpal of the right hand.

On January 3, a biopsy was taken from the right forearm. It was reported as showing skin, subcutaneous tissues, and venous thrombosis. The vein showed intimal thickening and scarring of the media. Diagnosis was phlebosclerosis.

On January 5, the venous pressure was measured and found to be 220 mm. in the right arm compared to 160 mm. in the left. There was no essential difference in the venous pressure of the two hands.

*Presented at meeting of the Orleans Parish Medical Society, May 10, 1948.

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On January 8, a venogram was done and was reported to show normal patency of the superficial and deep veins. The axillary vein and its tributaries were nicely demonstrated. A zone of spasm was noted in the basilic vein at one point.

Blood drawn from each arm simultaneously showed, in one instance, a definite increased oxygenation on the right side as evidenced by its brighter red color. This test was repeated but the second time no difference could be detected grossly.

Using 1 per cent novocain, a right stellate block was done on January 15. The patient was completely relieved of his pain for the duration of the block.

Operation: On January 23, 1948, under pentothal, ether, and curare, the second and third thoracic sympathetic ganglia with the intervening sympathetic trunk and the rami connecting them with the second and third intercoastal nerves were removed. The posterior approach recommended by Smithwick was used for this preganglionic sympathectomy. A transient Horner's syndrome was noted for a day or two postoperatively, and atelectasis of the right upper lobe occurred but re-expansion was prompt and uncomplicated. The patient was discharged February 7, 1948, with complete relief of pain. The hyperesthesia and tenderness of the extremity had disappeared.

DISCUSSION

In my opinion, this case belongs to a group of symptom complexes which have been given various names and which on superficial examination may seem to be unrelated. The term used or coined has usually served to emphasize the symptoms or signs which were outstanding in the case described but all tend to have in common, pain, sympathetic disturbances, and trophic changes.

Pain tends to be causalgia-like, that is, burning in character. There may be paresthesia and areas of hyperesthesia or there may be hypesthesia. The sensory finding often do not fit known or recognizable neurological patterns. The sympathetic imbalance may be manifested in protean ways, and in different cases there may be diametrically opposite effects. Thus in one there may be increased temperature locally, while in another it may be decreased. Trophic changes may be predominantly in the bones as in osteoporosis or Sudek's atrophy, or there may be an atrophy of the subcutaneous tissues as in the case reported.

It is frequently difficult to explain the mode of origin of pain in these patients, as pain perception has both a psychic and a physiologic basis. Furthermore, opinions are in some instances controversial and answers to certain questions have yet to be supplied by the neurophysiologist. For instance, the question of whether or not the sympathetics carry afferent pain fibers is not yet decided. The consensus of opinion is that they do not, although Threadgill has recently presented evidence to the contrary. Time and space do not permit a consideration of the explanations which have been offered as to how a single stimulus or one of relatively short duration can result in indefinitely prolonged response. Livingston[1] in *Pain Mechanisms* has emphasized the importance of the internuncial pool, and that in thinking of response to sympathetic stimulation, we must get away from our classical conceptions of the spinal reflex or short neuron arc. Sympathetic response is diffuse, prolonged, and often delayed. Therefore, according to Livingston, it is possible to set up a self-perpetuating "vicious circle." Kolodny[2] has emphasized the far reaching effects of peripheral sympathetic stimulation by showing the changes in the pattern of the electroencephalograph which such stimulation provokes. He believes that many common clinical observations such as syncope on pleural puncture can be explained on reflex cerebral vasoconstriction resulting from a peripheral stimulus, and Volkmann's contracture on reflex arterial spasm. The field is open for fruitful speculation on problems of major clinical significance—peptic ulcer and hypertension for instance.

It should be emphasized that it is not being implied in the type of case under discussion that pathology exists in the sympathetic nervous system. Sympathetic fibers, possibly on the afferent, but certainly on the efferent side, are merely transmitting impulses in a pathological reflex arc. The relief of pain in thrombophlebitis by lumbar sympathetic block has been explained by Oschner and De Bakey as resulting from relief of vascular spasm.

If we accept the theory that pain in

these causalgia-like syndromes is mediated through a reflex arc, the most rational approach to treatment is an attempt to interrupt this arc at some point. The logical point of attack would seem to be at the site of injury. In some instances the resection of a neuroma in an amputation stump or a neurolysis in a case of causalgia does give relief, but often this relief is temporary or fails completely. If it is kept in mind that the reflex may be perpetuating itself in the central nervous system this is not surprising. Phantom limb pain, for instance may not be relieved by reamputation. Likewise peripheral nerve block, sympathetic block, or the injection of a trigger zone as advocated by Stiendler are well worth trying before resorting to sympathetic surgery. Injections should be repeated if temporary relief is obtained. It is difficult to explain how a single block or even repeated blocks could give relief except on the grounds that we are interrupting a vicious circle because the injection of novocaine, the effects of which last only an hour or so, could hardly be expected to change existing local pathology directly.

Admittedly these patients are sometimes hard to evaluate. Occasionally there is a constitutional inferiority. Frequently the initial trauma is minor. Often the neurological findings do not fit recognized patterns, and the question of compensation in cases of occupational trauma may arouse the suspicion of malingering. Unquestionably, however, some of these patients have, in the past, been branded as neurotics, malingerers, or addicts, when actually organic pathology existed, and pain could have been relieved.

CONCLUSIONS:

1. A case of reflex sympathetic dystrophy of an upper extremity, relieved by dorsal sympathectomy, is presented.

2. This case is considered to belong to a group, all of which have in common the presence of pain, evidence of sympathetic disturbance, and trophic changes.

3. The role of the sympathetic nervous system in these syndromes is touched upon.

4. Treatment should consist in correction of obvious local pathology, injections of trigger zones, peripheral nerve injections, sympathetic block, and physiotherapy when it can be tolerated.

5. Sympathectomy is a useful method of treatment in those cases in which sympathetic block affords only temporary relief.

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INJURIES OF PERIPHERAL VESSELS*

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SHREVEPORT

Isolated injuries of major vessels of the extremities are not particularly common in civilian life, although, often enough, serious extremity injuries have a complicating factor of impaired circulation. Such isolated injuries, moreover, are not too common in military surgery. De Bakey and Simeone[24] in a most excellent and thorough statistical study of some 2,400 World War II cases state that an over-all incidence of 1.4 per cent is fairly representative.

CLASSIFICATION

Injuries of peripheral vessels may be divided into two broad groups: (1) the nonpenetrating and (2) the penetrating. In the nonpenetrating variety are included (a) concussion or contusion of major vessels and (b) cold injury. In the second group of penetrating injury may be included: (a) complete severance of a major

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vessel, (b) "false" aneurysm and (c) arteriovenous fistula.

BASIC PRINCIPLES

There are three basic principles which, if borne in mind, are of prognostic, prophylactic and therapeutic value. The first of these is that *trauma is more serious if a vascular lesion pre-exists.* The most practical import of recognizing this fact, perhaps, is in the prevention of cold injuries, i.e., in having those individuals so predisposed to avoid exposure. The second basic principle is that *gangrene develops when there is a discrepancy between tissue demands and blood supply* (Figure 1). Bearing this in mind will prevent one from making such therapeutic blunders as increasing tissue demands by the use of a heat cradle when the blood supply is impoverished. The third basic principle, and certainly the most clinically important, is that *vasospasm is an invariable accompaniment of vascular injury* [6, 32, 36, 46, 48, 57, 68].

NON-PENETRATING INJURIES

Contusion: It is difficult to estimate how many instances of gangrene have resulted from simple contusion or concussion of a major vessel. We[3] have reported a case of thrombosis of the radial and ulnar arteries from pressure on the forearm during sleep and an instance where extensive gangrene of the hand developed from intense vasospasm following contusion of an extremity. Such instances are not too uncommon. As the result of injury to an extremity, an extensive and serious vasospasm may result and/or there may be actual thrombosis in a major vessel endangering part of the extremity. Such injuries, too, are frequent in extremity injuries primarily of the soft parts or of bone, and many account for the interference with circulation in such injuries. The most important clinical point is to recognize the associated vasospasm and early institute measures to relieve it.[37]

Cold injury: There are several types of injury included in this category: Chilblains (pernio), frost bite, trench foot, immersion foot and high altitude frost bite. Chilblains is quite similar to trench foot in its pathogenesis and pathology[50] but is likely to oc-

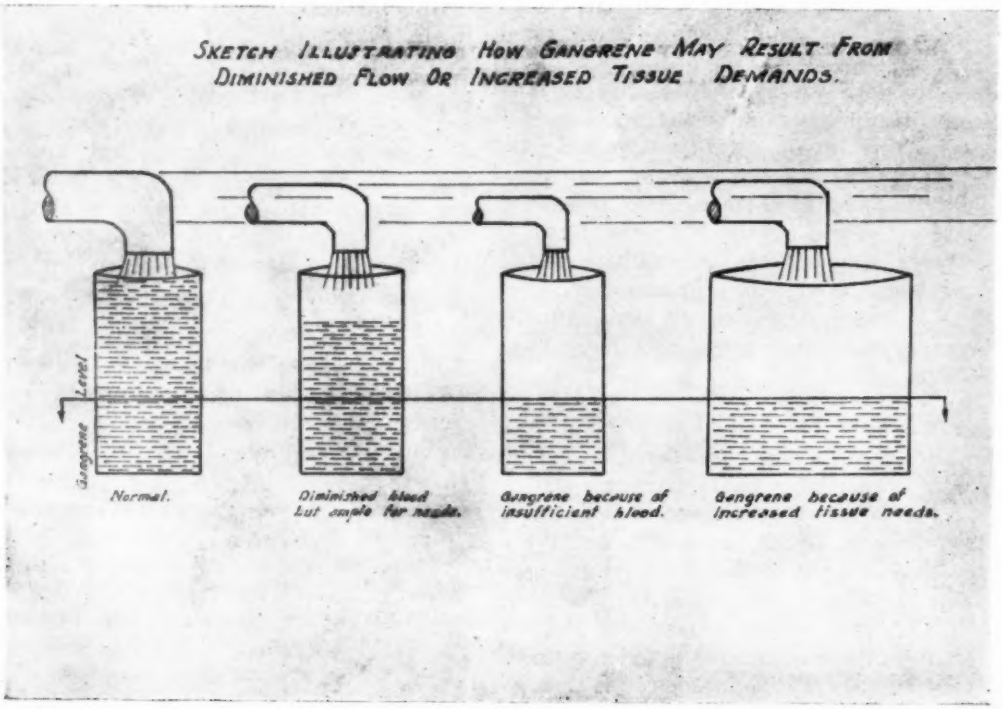
SKETCH ILLUSTRATING HOW GANGRENE MAY RESULT FROM DIMINISHED FLOW OR INCREASED TISSUE DEMANDS.
Gangrene Level
Normal.
Diminished blood but ample for needs.
Gangrene because of insufficient blood.
Gangrene because of increased tissue needs.

Fig. 1.

cur only in susceptible individuals, with even moderate exposure to cold. The skin appears to be chronically inflamed, is cold to the touch and is moderately painful.[67] "Frost bite", according to Lewis,[50] should be reserved for actual solidification (freezing) of tissues. Trench foot is the result of exposure of the feet to coldness and dampness. Immersion foot is the result of prolonged exposure of the extremities to cold water. High altitude frost bite is the condition which develops from the extreme cold, stasis and anoxia present in flying at high altitudes.

Cold injury is, undoubtedly, the most frequent type of vascular injury in both civilian and military practice. For example, in the 5th Army, for the week ending January 14, 1945, 46 per cent of all casualties were "trench feet"[19] and frost bite was a constant hazard of high altitude flying.[23]

It is not definitely proven that the pathology in all types of cold injury is identical, but there is definite indication that it is. Latimer[15] described the pathology of trench foot and divides it into three states: (1) the pre-hyperemic or ischemic state, (2) the hyperemic or inflammatory state and (3) the post hyperemic stage. Probably the best conception of the underlying pathologic physiology has been that of Loewe, Lange and associates[40, 41, 42, 51]. It has long been realized that both vasospasm and thrombosis were present, but the *time lag* in the development of thrombosis and its clinical significance has only recently been appreciated. Thus Loewe, *et al* have shown, by animal and human experiments on frost bite that the following four stages can be noted: (1) immediate slowing of the blood due to arteriolar (vasospastic) constriction; (2) anoxia, resulting in increased capillary permeability; (3) one-half hour after exposure this anoxia and permeability cause vasodilatation and swelling—the red cells are clumped because of hemoconcentration, but no clot is apparent until (4) after 72 hours. These same changes are presumed, but not proven, to occur in other types of cold injury. This conception has resulted in a new therapeutic approach (v.i.)

Undoubtedly, the prevention of these cold injuries is far better than trying to treat them once they have occurred. The best prophylactic measure would be the avoidance of exposure by susceptible individuals, that is, those with cold sensibility or peripheral vascular disease. Brahdy[10, 11], in a study of frost bite among New York City employees, found that a temperature below 14° F. constituted an industrial hazard, particularly if the individual already had diabetes or arteriosclerosis. Patterson and Anderson[62], as the result of study of exposure cases on Attu, recommended eliminating from exposure, as far as practical, those with previous foot or leg injury, those with signs suggesting vasomotor instability or peripheral vascular disease, and those with foot infections. In addition to eliminating, as much as possible, susceptible individuals from exposure, the use of protective clothing and devices, combating of stasis and dampness by exercise, massage and change of socks were used in an effort to reduce the number of cold injuries.

Once symptoms have been induced by exposure to cold, it is extremely important that the individual be removed from the low temperature. As Brahdy[10, 11] pointed out in his reports, an important prognostic point is the length of exposure after the first symptom: the sooner the exposure is eliminated, the less likelihood there is of dire consequences. In handling trench feet and similar cold injuries during World War II, the results were disappointing. Often it was patently impossible to remove the exposed individual promptly. Once he was hospitalized, the usual therapy was bed rest, preservation of heat by wrapping in sheet wadding, in some instances sympathetic procaine block, prevention and treatment of infection, relief of pain. Once demarcation had occurred, in those cases progressing to gangrene, amputation was necessary. Many cases never developed gangrene, but retained painful, discolored, disabling extremities[45, 61].

The studies of Lange and his associates[40, 41, 42] may well alter our results in the

treatment of cold injury, particularly the frost bite seen in civilian practice. Based on their experimental and clinical observations (v.s.) they conclude that if heparin is administered within forty-eight hours of exposure, the occlusive red-cell masses do not form, and subsequent gangrene is thus prevented. They furthermore confirmed previous observations that both cooling and warming are harmful and that maintenance of normal room temperature is the safest procedure. In the exudative stage, sympathetic block may help by causing complete, rather than localized vasodilatation, thus increasing tissue circulation, relieving anoxia, increasing lymph circulation and reducing the edema. In those cases developing gangrene, one may be quite conservative, removing only the dead tissue.

PENETRATING INJURIES

Severance of a main artery: As previously noted, DeBakey and Simeone[24] in a study of 2,400 World War II cases found an overall incidence of major arterial injury to be 1.4 per cent. Odom[59], who was surgeon to the 3rd Army, reported that of 92,000 casualties treated by 3rd Army installations only 837 or 0.9 per cent were classifiable as primarily vascular. This does not mean that injuries to major vessels were not more frequent, but that extensive soft tissue damage or badly compounded fractures made the arterial injury not the primary diagnosis.

In cases in which a main artery is severed, the purposes of treatment are twofold: (1) control of the hemorrhage, combating shock, prevention of infection and (2) preservation or increasing the blood supply. In carrying out these purposes there is the tourniquet, for temporarily controlling the bleeding; blood and blood fractions for shock; debridement, chemotherapy, antibiotics and immunotherapy to prevent infection. The role of refrigeration is debatable. Holman[37] suggested the use of ice bags in front line casualties, (an impracticable idea for several reasons) on the theoretical grounds that shock would be reduced, infection held in abeyance and the tourniquet could be left on safely for a longer period. Likewise, Crossman and Allen[21] do not agree with Standard[72] that there is damage to tissue by prolonged exposure to refrigeration temperatures. However, as De Bakey and Simeone[24] point out, the "wisdom of actual refrigeration is still open to question for any purposes except to control infection and diminish lymphatic absorption from an infected limb prior to amputation, or to permit amputation without anesthesia in an aged and debilitated subject." Despite Crossman and Allen's enthusiasm, there is evidence[12, 17, 43, 44] demonstrating that refrigeration impairs wound healing, wound infection progresses more rapidly once refrigeration is stopped, and nerve damage is likely to occur.

Sympathetic interruption to relieve vasospasm is well entrenched in the treatment of vascular injuries. It has been amply shown that vasospasm is an invariable accompaniment of vascular injury and its relaxation by procaine sympathetic blocks was frequently used to help in the development of collateral circulation and the preservation of the part. Published figures[24] might indicate that poorer results were obtained from sympathectomy than from sympathetic block, but this is probably explained on the basis of the fact that sympathectomy was often only used as a last resort and in practically unsalvageable cases. Though it is difficult to prove, on the basis of published data, there is reason to believe that sympathetic block or sympathectomy may play an important part in the preservation of a vascularly endangered extremity.

In the management of severed major arteries, both venous and arterial ligation have been practiced. Holman and Edwards[38] showed experimentally that if the vein is ligated higher than the artery, both the blood pressure and the blood flow are increased in the distal stump of the ligated artery. The experimental observations of Brooks and his associates[13, 14, 15] have indicated its value, though Leriche[47] believes the value derived from vein ligation is the result of relaxation of vasospasm for which we have more direct means of relief. De

Bakey and Simeone[24] give an extensive review of the pros and cons of this procedure and conclude that "ligation of the concomitant vein furnishes no protection whatsoever against the development of gangrene after acute arterial occlusion and ligation in battle casualties." Whether it is of value in civilian injuries is equally unlikely.

Arterial ligation was the most practical and widespread procedure practiced during World War II. This was not only because of the frequent lack of time for a more lengthy procedure, but often it was the only type of repair possible. As has been emphasized[24], one must clearly distinguish between ligation in acute arterial injuries and ligation in a complication, such as an aneurysm; in the latter instance there have developed collaterals not present in acute lesions.

Theoretically, arteriorrhaphy, either with some appliance such as the Blakemore tube or by simple suture repair, should be the best procedure. Yet, statistically at least, simple ligation in acute injuries does not appreciable affect the incidence of gangrene. It should be remembered that there are certain "critical arteries", or more specifically, gangrene is more likely to follow ligation of some arteries than of others. Thus ligation of the femoral artery will result in 80 per cent incidence of gangrene, as compared to 50 per cent following ligation of the axillary artery, 35 per cent following ligation of the brachial artery and 71 per cent following ligation of the popliteal[70]. This factor of varying importance of different arteries, plus varying degrees of associated injury, plus the fact that exactly comparable circumstances rarely exist in two series being treated, one by ligation, the other by repair, make it impossible to accurately interpret comparative figures. Admitting the inaccuracies, little confidence can be based on the figure of Smith[70] and of Rose, Hess and Welch[66], showing 50 per cent gangrene in cases of ligation as compared to 44 per cent after some type of anastomosis, or of Odom[59] showing 50 per cent gangrene following ligation as compared to 53 per cent following an anastomosis, or of De Bakey and Simeone[24], with 48.9 per cent gangrene after ligation compared to 44.4 per cent following repair. Certainly in civilian practice the injuries are likely to be less severe, the cases are likely to receive definitive treatment earlier and under less harried circumstances, and it should be possible to do reparative procedures more and more frequently either by simple suture, by vein graft or by some type of tube repair. The addition of heparin[58] to the therapeutic armamentarium may prove helpful, but this remains to be seen. In ligation of a main artery two things should be borne in mind: First, the ligation should not be in continuity, but rather should the vessel be severed between ligatures[27], thus helping to relieve vasospasm by doing, in effect, a periarterial sympathectomy. Second, relaxation of vasospasm by procaine block or sympathectomy should be a *sine qua non*.

"False" aneurysm: This is in reality, a pulsating hematoma and results when there is an incomplete severance of an artery and the development of a hematoma in the soft parts surrounding it. Its dangers are twofold: First, it may rupture to the outside, producing secondary hemorrhage, or second, it may, in enlarging, further damage the peripheral arterial supply or damage nerves[35]. These should be recognized and treated early, by evacuation of the clot, ligating or repairing the vessel, as circumstances permit, and interrupting the sympathetic flow. If delay has occurred before the "false" aneurysm is recognized, and it is not enlarging and there is no danger of external rupture, then delay, to encourage the development of collaterals is justified.

Arteriovenous Fistula: Such a lesion obviously occurs when an artery and its concommitant vein are perforated. It is often not immediately diagnosed and there is little danger from secondary hemorrhage or soft tissue pressure, as the artery "decompresses" itself into the vein. We[1] have pointed out that the clinical effects of peripheral arteriovenous fistula can be divided into (1) the *local effects* (pulsating

tumor, bruit, and thrill), (2) the *peripheral effects* (admixture of arterial and venous blood, dilation of peripheral veins, etc.) and (3) the *systemic effects* (increase in pulse rate, increase in systolic pressure, decrease in diastolic pressure, increase in blood volume, late cardiac damage). Similarly, we have emphasized that the diagnosis is not difficult if it is suspected. The diagnostic points to be recalled are: (1) The history of a typical penetrating trauma; (2) the presence of a pulsating tumor with characteristic thrill and bruit; (3) dilated veins distal to the fistula with an increase in venous and oxygen tensions; (4) trophic disturbances distal to the fistula—edema, pigmentation, ulceration; (5) the Nicoladoni-Israel-Branham phenomenon. An arteriogram is frequently confirmatory.

The treatment of arteriovenous fistula should be either some type of aneurysmorrhaphy or quadruple ligation. In most instances, the quadruple ligation has been used and is quite satisfactory[63, 65]. In either type of treatment, preliminary development of collaterals, by fistula compression and sympathectomy are valuable adjuncts. Undoubtedly, the various types of anastomatic repairs, with or without heparin, will be increasingly useful.

SUMMARY AND CONCLUSIONS

1. Injuries of peripheral vessels may be divided into (a) non-penetrating and (b) penetrating varieties.

2. Non-penetrating injuries include (a) concussion and contusion and (b) cold injuries.

3. Penetrating injuries include (a) severance of an artery, (b) "false" aneurysm and (c) arteriovenous fistula.

4. Three basic principles in the management of peripheral vascular injuries are: (a) trauma is more serious if a vascular lesion pre-exists; (b) gangrene develops whenever there is a discrepancy between tissue demands and blood supply and and (c) vasospasm is an invariable accompaniment of vascular injury.

5. Cold injuries are the most frequent type of peripheral vascular injury and its several varieties are chilblains (pernio), frost bite, trench foot, immersion foot and high altitude frost bite.

6. In penetrating wounds in which a main artery is severed, the purpose of treatment is twofold: (a) control hemorrhage, combat shock, prevent infection and (b) preserving or increasing the blood supply. In most instances, ligation has been used but arteriorrhaphy will become increasingly more useful.

7. "False" aneurysm has two dangers: (a) it may rupture to the outside resulting in a secondary hemorrhage, or (b) by enlarging it may compress collaterals or nerve filaments. Therefore its early correction is usually indicated.

8. An arteriovenous fistula has local, peripheral and systemic effects which make its detection fairly simple. Such fistulae should be corrected, as soon as collaterals have been developed, either by arteriorrhaphy or quadruple ligation.

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DISCUSSION

Dr. Isidore Cohn (New Orleans): Dr. Abramson's paper gives evidence of careful survey of the literature and excellent judgment. Under the heading of basic principles he mentions prophylaxis. Very properly he said trauma is more serious if a vascular lesion preexists. In a preventive way much can be done by proper attention to the lower extremities in the aged under ordinary conditions. I mean by that long before they have had an accident. In discussing injuries to the main arteries, Dr. Abramson mentions control of hemorrhage and shock, the prevention of infection and preserving or increasing blood supply. He expressed the hope that arteriorrhaphy will be used more frequently in the future. This presupposes that the facilities for suture and anti-

coagulants will become more generally available in our hospitals. It certainly is a development to be hoped for. With ever widening interest in vascular surgery, the principles for safer suture methods as well as so-called nonsuture methods are being offered for preserving the vascular channels after injury. He approached the early adequate management of damage to particular arteries—it is necessary that the surgeon know anatomy. If there are any former students of the late Dr. John B. Elliot they will remember his saying, "Gentlemen, remember anatomy." Direct anatomical approach saves time and structures. Too much has been covered to attempt a detailed discussion. Fundamental principles have been given by Dr. Abramson in concise manner. I would like to congratulate him on his splendid presentation.

—o—

THE CORRELATION BETWEEN CLINICAL ESTIMATION AND LABORATORY DETERMINATION OF FUNCTIONAL PULMONARY CAPACITY*

LEO E. JOHNS, JR., M. D.
AND
JOHN H. SEABURY, M. D.
NEW ORLEANS

There have been many recent advances in the field of thoracic surgery. These advances have been due principally to perfection of newer and more versatile surgical technics, the development of satisfactory methods of anesthesia, the advent of the antibiotics which allow more successful combatting of infections, and the establishment of sound physiologic methods of pre- and postoperative care. As a result of these advances there has been a wide extension of general indications for major thoracic surgical procedures.

Any procedure which irreversibly decreases the functional capacity of one or both lungs should be undertaken only when the responsible physician believes that sufficient pulmonary function will remain to support the patient comfortably when he engages in the minimum amount of activity necessary to life in bed. This simple principle has become more apparent and important as the horizons of thoracic surgery have been extended. If the total functional capacity be known, together with the distribution of this function between the two lungs, the surgeon can approach intelligently and with confidence the decision of operability for each patient. When this decision must be made on the basis of clinical judgment alone, some patients, even though they survive the surgical procedure, are left with pulmonary function so impaired that the result is complete invalidism, or at times an actual shortening of life to a degree not to be expected from the natural history of the pulmonary disease. Consequently, the study of pulmonary physiology in man has been accelerated and technics have been developed which permit a fairly precise determination of lung function.

Complete evaluation of pulmonary function, omitting the consideration of interrelated cardiac function, consists essentially of a consideration of two phases. First, the *ventilatory function*, or the ability of the anatomic apparatus to serve as a bellows in mechanically moving air in and out of the lungs. This depends on the action of the intercostal musculature and the diaphragm, the size, shape, and fixation or lack thereof of the costal cage, the presence or absence of pleuritis, and bronchial obstruction, as well as on the results of any actual involvement of lung tissue by a pathological process. Second, the true *respiratory function*, or the diffusion of gases across the alveolar membrane which depends on the conditions present at the alveolar epthelium and small pulmonary vessels on the one hand and on the conditions present within the oxygen-carbon dioxide transport system (red blood cells and plasma) and tissue acceptor system on the other hand The strictly pulmonary side of respiratory function is diminished by pulmonary fibrosis, edema, accumulation or organization of exudates, and changes in the walls of the pulmonary blood vessels

In intrathoracic disease there is almost

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always some impairment of both of these functions, and they are closely interrelated but not directly proportional one to the other. It is obviously necessary for ventilation to be adequate in order to obtain the required gaseous diffusion, but adequate ventilation does not necessarily imply sufficient respiration. Diffusion of gases must be adequate for ventilation to be effective, no matter how efficient ventilation per se may be; and at times impairment of ventilation of even marked degree may be compensated by the efficiency of gaseous diffusion in the localized areas which are unaffected by disease processes. These two functions, although sequential and superficially inseparable, often are not impaired in any direct proportion, and evaluation of one without adequate knowledge of the other can lead to grossly erroneous assumptions.

The logical approach to the evaluation of pulmonary function, therefore, depends on the proper correlation of these two phases of "respiratory" physiology. Ventilatory function may be measured by the determination of pulmonary volumina, the vital capacity and its various components, and by the determination of the maximum breathing capacity over a given, arbitrary, period of time, either after graded exercise or during forced voluntary hyperventilation. Respiratory function may be evaluated by analysis of gaseous exchange, either by direct analysis of alveolar air and blood gas tensions. Evaluation of total pulmonary function from measurements of ventilatory function alone often fails to give a true picture of the existing situation.

In the majority of patients with thoracic disease estimation of the total ventilatory and respiratory function, together with study of the history, physical examination, and roentgenograms, allow prediction of the effects of the contemplated surgical procedure upon these functions. However, these studies alone may be misleading and will result in false assumptions in approximately 15 per cent of patients evaluated by these methods only. Previous lung disease may have left residual damage which is undetectable by x-ray, or the effects of the present disease are over-evaluated. By clinical judgment alone, one is unable to determine the distribution of the various functions between the two lungs of these patients. In such cases it would seem necessary to employ a technic of objectively quantitating this distribution. Bronchospirometry, a procedure at least no more dangerous to or uncomfortable for the patient than simple bronchoscopy, offers such a method.

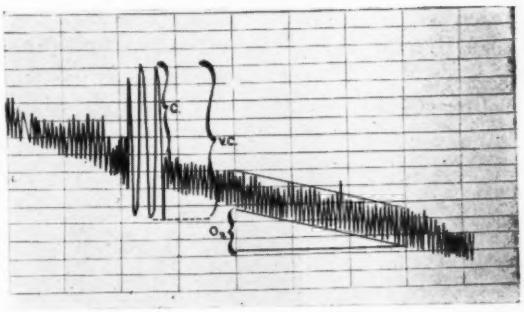

Fig. 1. Sample graphic record of total pulmonary function obtained with modified Benedict-Roth spirometer. By proper calibration the pulmonary volumina, minute volume of respiration, and respiratory rate can be determined. O_2 consumption can be estimated from the slope of the curve.

In doing bronchospirometry routinely in the Lung Station of the Charity Hospital, we find that the data so obtained are usually merely a corroboration of the status of function predicted by our clinical evaluation, especially when supplemented by careful fluoroscopy. However, there are surprises which throw new light on surgical risk. It is not possible to know just where or in what patient we will meet with unexpected results. As previously stated, this group comprises about 15 per cent of all patients studied.

Most of our work with the double-barrel bronchial cathether has been on patients with tuberculosis, but the method is equally applicable and pertinent to all other surgical diseases of the chest. It should be stated that the results obtained by this method do not represent absolute determinations of pulmonary function due to the factor of stenotic resistance imposed by the catheter;

they are nevertheless reliable indices of the relative function between the two lungs, the factor of resistance being equal bilaterally. This equality of resistance permits one to express the values for each lung as a percentage distribution of the total pulmonary function.

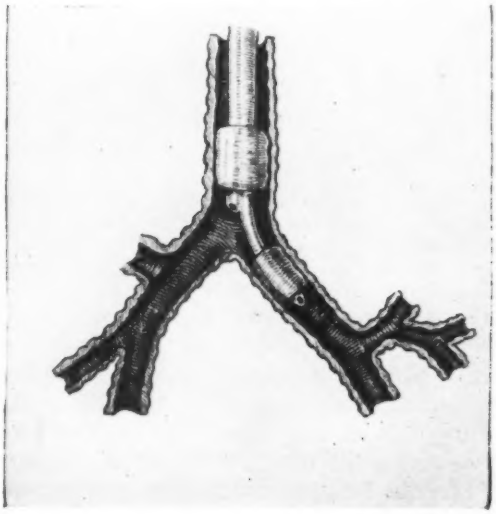

Fig. 2. Zavod catheter in proper position in trachea and left main stem bronchus for broncho-spirometric determinations. By connecting ends of catheter to separate recording spirometers simultaneous tracings may be obtained from right and left lobes. Tidal ventilation and O_2 consumption may be measured as in Fig. 1.

The following illustrations and brief case summaries are examples of the discrepancy which may exist between clinical estimation and laboratory determination of pulmonary function.

Figure 3 is the roentgenogram of a patient with tuberculosis who gave a history of pleuritic pain in the right chest for four months. After the diagnosis was established, right pneumothorax was instituted but abandoned in six months due to numerous adhesions. One year later the studies illustrated in Figure 4 were done. Note the large cavity in the right upper lobe, the small area of pneumonitis in the left mid-lung zone, the absence of any definite evidence of pleuritis on either side, and the lack of any mediastinal shift. Total studies

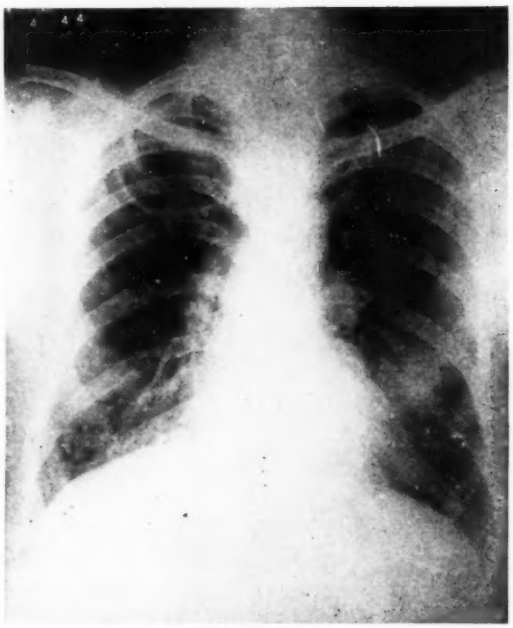

Fig. 3.

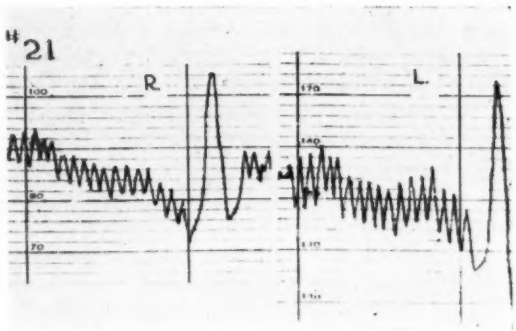

Fig. 4.

revealed moderate impairment of vital capacity (3.05 liters). It might be surmised from the history and x-ray that such reduction is largely due to the right-sided disease. Yet Figure 4 reveals that the ventilatory ratio is $\frac{\text{Right}}{\text{Left}} = \frac{60.6}{39.4}$. To explain this apparent discrepancy it should be remembered that, costal and diaphragmatic movement being sufficient, cavities which freely communicate with bronchioles may be ventilated freely. The previous pleuritis would appear to be less detrimental to ventilation than would be suspected. Also, since these

results are relative, it must be concluded that impairment of total function is due to increased ventilation on the left side to a great extent. This is probably due to previous disease, a history of which was not obtainable.

The respiratory ratio is $\frac{\text{Right}}{\text{Left}} = \frac{37.1}{62.9}$. Oxygen consumption on the right is markedly decreased in relation to ventilatory ability. This can be explained by impaired gaseous diffusion around the cavity, and the increased dead space produced by the cavity.

Figure 5 reveals a right-sided hydrothorax following therapeutic pneumothorax. Vital capacity was moderately reduced (2.74 liters). Figure 6 shows tracings

Fig. 5.

from this patient. The ventilatory ratio was $\frac{\text{Right}}{\text{Left}} = \frac{41.4}{58.6}$. The respiratory ratio was $\frac{\text{Right}}{\text{Left}} = \frac{53.3}{46.7}$. The function on the obviously diseased right side is surprisingly good. Of as great importance is the fact that, since total function shows impairment, the function of the "good" left side is surprisingly poor.

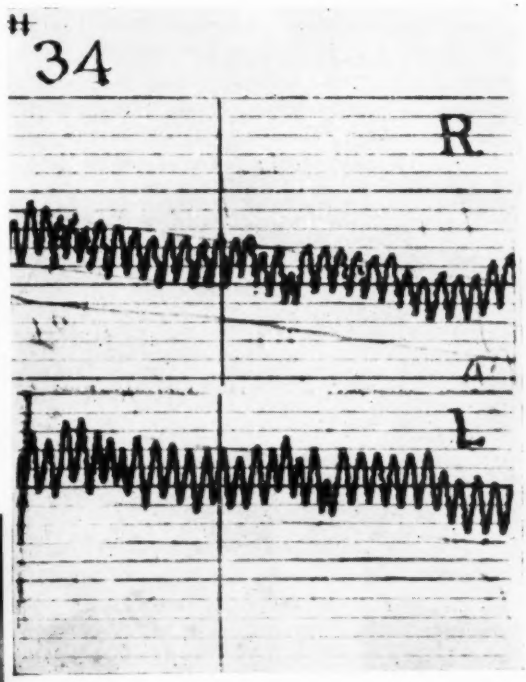

Fig. 6.

Figure 7 presents a picture of residual

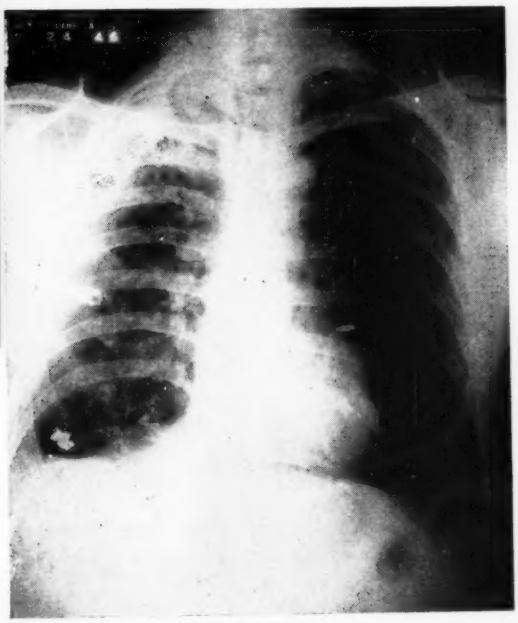

Fig. 7.

pathology following right pneumothorax complicated by effusion and converted to right oleothorax for one year. In this patient there also had been an unsuccessful attempt to establish pneumothorax on the left, suggesting pleural symphysis on that side.

Figure 8 illustrates bronchospirometric

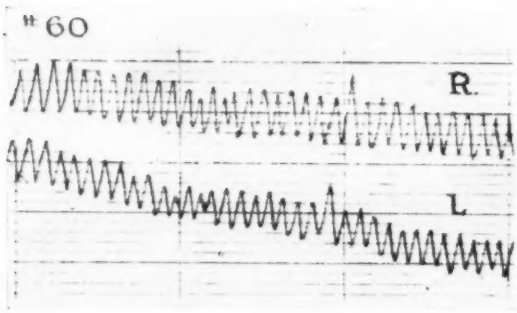

Fig. 8.

tracings on this patient. The vital capacity here was 2.74 liters. The ventilatory ratio was $\frac{\text{Right}}{\text{Left}} = \frac{56.4}{43.6}$. The respiratory ratio was $\frac{\text{Right}}{\text{Left}} = \frac{33.3}{66.7}$. The normal ventilatory ratio was unexpected as was also the discrepancy between ventilatory and true respiratory function on the right side. Ordinarily, the pleural effects of oleothorax are considered to reduce the ventilatory much more than the respiratory function. However, the inapparent pleural symphysis on the left so impaired the ventilatory function on this side that the percentage distribution of ventilatory function between the two sides remained normal.

Figures 9 and 10 are x-rays of a patient

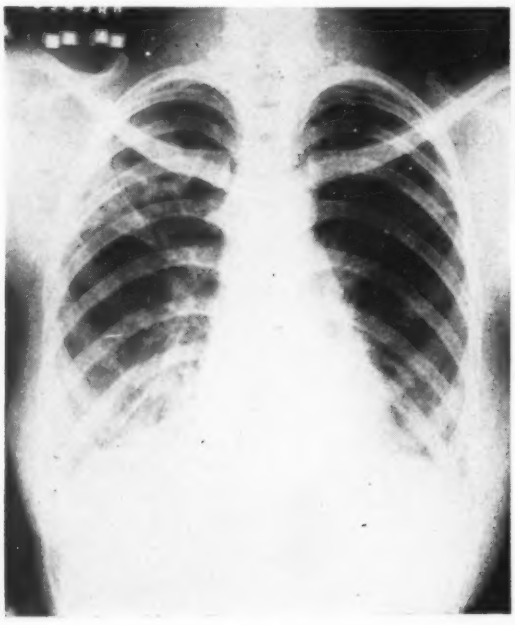

Fig. 9.

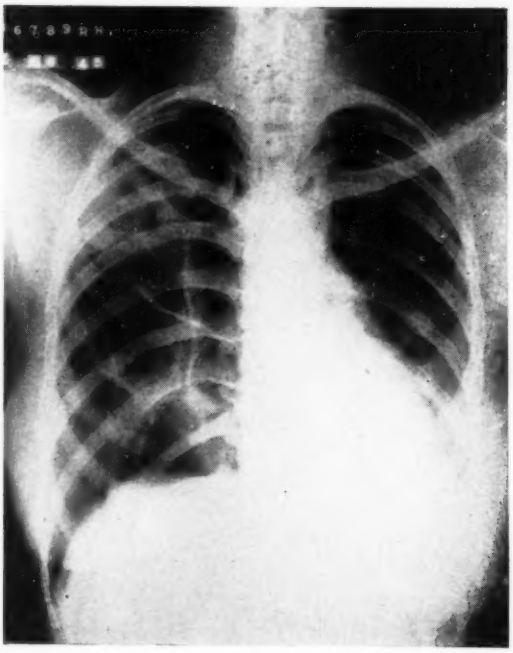

Fig. 10.

who had a pneumothorax started and abandoned on the right side before admission. Lung studies revealed the total pulmonary function to be too poor to consider collapse procedures as extensive as thoracoplasty so right pneumothorax was again established (Figure 10). Note the adherent areas with partial collapse of lung on the right and the mediastinal shift with compression of lung on the left. Bronchospirometry (Figure 11) revealed that the ventilation and oxygen consumption were significantly better on the right than on the left.

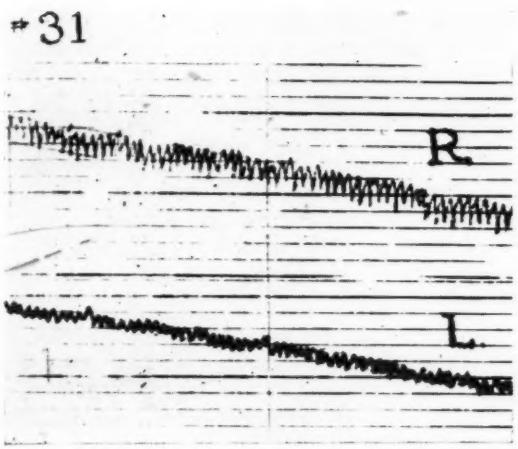

Fig. 11.

Ventilatory ratio: $\frac{\text{Right}}{\text{Left}} = \frac{68.8}{31.2}$.

Respiratory ratio: $\frac{\text{Right}}{\text{Left}} = \frac{52.4}{47.6}$.

Reduction of function on the left side was probably due to mediastinal shift with tracheobronchial compression, atelectasis, and retention of secretions.

There are similar records of many other cases in which fairly large errors of judgment would have been made had we been dependent upon clinical evaluation and x-ray diagnosis without the added knowledge revealed by bronchospirometry.

In conclusion, it should be emphasized that:

Candidates for thoracic surgery should be evaluated in the light of both ventilatory and respiratory function. Estimation of vital capacity alone is not sufficient and may be grossly misleading.

In about 15 per cent of such patients clinical judgment and total studies are insufficient to estimate the distribution of these functions between respective sides. At present this significant percentage of error can be avoided only by routine bronchospirometry. Consequently, the thoracic surgeon can receive great help from bronchospirometry both in the selection of suitable patients and in choosing the procedure most likely to effect a cure without producing serious respiratory embarrassment.

The authors acknowledge with thanks permission of the American Review of Tuberculosis to use in this paper illustrations which had appeared in the Am. Rev. Tuberc. 57:254, March 1948.

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CLINICAL EXPERIENCES WITH THE Rh FACTOR IN OBSTETRICS*

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AND

J. A. KING, M. D.

NEW ORLEANS

The Rh factor was discovered by Landsteiner and Wiener[1] in 1940, while searching for an explanation of transfusion reactions in patients receiving blood which was compatible by all known tests. It was soon found that this factor is concerned in the causation of erythroblastosis fetalis, which has been recognized as a clinical entity for years. Subsequent studies showed that fetal hydrops and icterus neonatorium were also due to Rh incompatibility, and that these three conditions are simply variations of the same disease.

Many thousands of tests have shown that approximately 85 per cent of whites have the Rh factor, and are designated Rh positive. Negroes, Chinese, and American Indians show a much higher percentage (Table 1 from Levine). Those individuals not having the Rh factor are designated Rh negative.

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*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

TABLE 1
TESTS WITH ANTI Rh_0 SERUM

Race	Number Tested	+ %	— %	Incidence of Erythroblastosis Fetalis %
Chinese	150	99.3	0.7	Very rare
White	234	85.0	15.0	2.1
Negro (Levine)	264	95.5	4.5	....
Negro (Landsteiner & Wiener)	113	92.0	8.0	0.7
American Indian	120	99.2	0.8	7

The Rh factor is not a single entity, but a complex one. Thus, another testing serum gives 70 per cent positive reactions (Rh^1), and still another 30 per cent (Rh_0). Some patients react to all three sera. The 85 per cent serum is the one generally employed. Elaborate studies have demonstrated the existence of other rare subgroups. It has also been found that sensitization has been caused occasionally by blood group incompatibility. For practical purposes, we are concerned chiefly with patients tested with the 85 per cent serum.

With 15 per cent of all females being Rh negative, we would expect to find a smaller percentage in married women. Thus Halperin, Jacobi, and Dubin[2] found 66 Rh negative women in 500 pregnant patients (13.2 per cent). Levin, Katzin, and Burnham[3] estimated that in 9 to 10 per cent of white marriages there will be the union of an Rh negative woman and an Rh positive man. In our series of 569 tests, there were 98 Rh negative women, a percentage of 17.3. Of the 74 husbands of these Rh negative women who were checked, 62 were positive and 12 were negative. The 24 not tested were nearly all husbands of primigravida.

However, the development of erythroblastosis in the baby occurs in only a small percentage of these marriages of Rh negative women to Rh positive men, and the reason is clear when the mechanism of the development of the condition is considered. If the fetus is Rh positive, some of its blood may in some way pass the placental barrier and get into the maternal circulation. This would produce sensitization of the mother to the Rh factor which she does not possess. Levin[4] believes that only .13 cc. of Rh positive fetal blood is needed to produce this isoimmunization in the mother. Once this occurs, anti-Rh agglutinins are formed in the maternal blood stream. This development is gradual; the transfer of fetal blood probably occurs rather late in the first pregnancy, so that only isoimmunization is produced, and no antibodies are formed. In the next pregnancy, if the fetus is Rh positive, antibodies may be produced by the mother, and may pass back transplacentally to the fetus, producing destruction of the Rh positive red cells (carrying the Rh factor), with a resulting overstimulation of the blood forming organs, and an outpouring of immature red cells. This may not occur until the third or fourth pregnancy, or it may never happen in some marriages.

This sensitization may be produced, and has been produced in several patients coming under our observation, by transfusion of an Rh negative woman years before marriage with Rh positive blood. Levin and Waller[4] believe that intramuscular injection of blood in newborn female infants may have produced isoimmunization in some patients with no history of transfusion. In such patients, the first baby (and all subsequent ones), may be erythroblastotic. As mentioned previously, some cases of sensitization may be due to Rh subtypes, blood group incompatibility, or to the rare Hr factor. These are so infrequent that they do not give us much concern.

Once the state of isoimmunization is produced, it persists for the life time of the woman (Levin).[5] No method of desensitization has yet been devised. Ethylene disulfonate was used in 3 cases with apparently satisfactory results,[6] but was found to be of no avail in a patient treated by us. The fetus is not affected until late in pregnancy, as the antibodies usually develop after the fifth month, and the titer tends to rise as pregnancy advances. Hence, the Rh factor is not concerned in the causation of abortion.

It is therefore important to test all obstetrical patients for the Rh factor. If

there is no history of transfusion, the testing may be omitted in primigravida, but it should be done if transfusion becomes necessary. All multigravida should be tested, if possible. When the patient is found to be Rh negative, her husband and the child or children already born are tested also. If the husband is negative, there is no possibility of sensitization, as his children will all be negative. If the husband is positive, the wife should be tested for antibodies each month after the fifth month. If none are found up to the end of pregnancy, the prognosis for the child is good. However, in one such patient, the infant was rather severely affected, and antibodies were found in the maternal and fetal blood only after delivery. If antibodies are found, and the titer increases, the prognosis is guarded, and is worse in case blocking antibodies alone are present. About 50 per cent of these Rh positive husbands are heterozygous, in which instance there is a 50 per cent chance that the infant will be Rh negative, and hence will not be affected unless sensitization to one of the rare factors occurs. Tests of the children already born will help in settling this point.

If antibodies are found, and the titer is rising, it would appear best to remove the child from the unfavorable environment before term, either by induction of labor or by Cesarean section. However, these procedures have not proved of much value, at least in our hands. The hazards of prematurity are added to the damage already done by the sensitization. Still, such premature delivery is worth while if the antibody titer is very high. After delivery, it is advisable to transfuse the baby at once if its condition is not good, especially if the amniotic fluid has a yellowish tinge. Replacement transfusion is of value in severely ill babies of the icteric or anemic type. No treatment is of any value for babies of the hydropic type. If there is little or no clinical evidence of erythroblastosis, a blood count should be done at once, and transfusion of 30-40 cc. performed if indicated. These transfusions should be repeated as often as the blood count requires. In one instance the baby was transfused 13 times over a period of six weeks. Only compatible Rh negative blood is used.

As stated above, of our 98 Rh negative patients, 12 are married to Rh negative men, and hence no sensitization is possible. Twenty-four husbands were not tested, their wives being primigravida with no history of transfusion. Hence, we have under consideration 62 Rh negative women with Rh positive husbands. Of these, in 43 instances Rh complications were possible. In 10 patients, the Rh factor did cause serious trouble, and 6 of these women lost a total of 8 babies (two women lost 2 each). These and other details are shown in table 2. The details of these 10 cases and of 6 others of interest are listed below in the case reports.

TABLE 2

TOTAL NUMBER OF WOMEN EXAMINED—569

Rh positive women		471	82.7%
Rh negative women		98	17.3%
Husbands of Rh negative women:			
Rh negative		12	
Not tested		24	
Rh positive		62	
Of the 62 Rh negative women married to Rh positive men:			
I. Set-up present for potential trouble		43	69.4%
a. Trouble occurred	10		
b. No trouble	24		
c. Not delivered yet	8		
d. Result of pregnancy not known	1		
II. Set-up not present for potential trouble		19	
a. Delivered without trouble	16		
b. Not delivered yet	3		

CASE REPORTS

Case No. 1. Mrs. V. C.

1st Pregnancy—Cesaeran Section eight months, placenta previa. Baby lived forty-four hours. Mother had one transfusion. Rh of donor and husband (first) not known.

2nd Pregnancy—second husband, cesarean section December 14, 1947. Wife Rh negative, husband Rh positive. Blocking antibody 1:16. Two doses ethylene disulfonate. Baby 5 lb. 14 oz., replacement transfusion. Lived.

The value of ethylene disulfonate in this case is questionable.

Case No. 2. Mrs. C.

1st Pregnancy—Delivered May 4, 1934—Girl Rh positive.

2nd Pregnancy—Delivered March 19, 1943—Spontaneous abortion.

3rd Pregnancy—Delivered October 3, 1944—Girl Rh positive—Normal.

4th Pregnancy—Delivered February 20, 1947—Girl Rh positive. Transfused. Mother had albumen antibodies and slight trace of blocking antibodies.

Rh factor not implicated in abortion. In this person, the difficulty did not show up until the third full term pregnancy.

Case No. 3. Mrs. R. C.

Wife Rh negative, husband Rh positive; homozygous.

1st Pregnancy—August 11, 1943—Girl.

2nd Pregnancy—Delivered November 2, 1947, premature, 3 lb. 13 oz. Mother had strong Rh agglutinins and moderate blocking antibodies. Baby in hospital two months, 13 transfusions. Baby's blood Rh negative at all tests, probably is really Rh positive, but negative reactions due to transfused blood.

February 23, 1948—Mother's blood showed Rh agglutinins 1:16, blocking antibody 1:32. Prognosis poor for another pregnancy.

Case No. 4. Mrs. A. D. Rh negative.

Ist Pregnancy 1935—Girl 9 lb. 14 oz., Rh positive; by first husband.

2nd Pregnancy 1937—Boy 8½ lb. Rh positive; by first husband.

3rd Pregnancy 1941—Boy 8 lb. 14 oz., Rh negative; by second husband.

4th Pregnancy 1945—Girl 7 lb., Rh positive; by second husband, lived five days; autopsy showed erythroblastosis.

5th Pregnancy 1946—Girl, term, Rh negative, section because of increasing titer of antibodies; residue from 4th pregnancy (?).

Second husband is hence heterozygous.

This represents a puzzler to us. The rising titer of antibodies suggested severe involvement and a bad prognosis, but the baby is Rh negative and unaffected, required no treatment.

Case No. 5. Mrs. DeB.

Transfused 1940, operation for ruptured appendix. Donor evidently Rh positive, not tested.

1st Pregnancy—1943, baby stillborn and macerated. Husband reported Rh positive, wife also reported Rh positive, later found to be an error, after second pregnancy had occurred, really Rh negative.

2nd Pregnancy—L.M.P. April 21, 1945. Antirodies found in July, increasing with subsequent tests. Section December 29, 1945, baby lived three days, transfused several times. Tests September 26, 1947 and February 19, 1948 showed antibodies persisting. Mother evidently sensitized by transfusions. Another pregnancy inadvisable.

Case No. 6. Mrs. R. F.

1st Pregnancy—1944, section, baby healthy.

2nd Pregnancy—1946, father Rh positive, mother Rh negative.

Antibodies found December 18, 1946. Section at eight and a half months, December 28, 1946. Baby lived three days, transfused several times. Autopsy showed hemorrhage into lungs, possibly due to overtransfusion.

Case No. 7. Mrs. H. S. F.

Wife Rh negative, husband Rh positive.

1st Pregnancy—1943, 6 lb. 4 oz. baby, healthy, Rh positive.

2nd Pregnancy — 1944, stillborn, edematous, autopsy showed erythroblastosis, baby's blood reported Rh negative. Probably an error. Baby dead four days before delivery, autopsy and blood test thirty-six hours after delivery. Antibodies (blocking) still present in mother's blood thirteen months after delivery.

3rd Pregnancy—1948, expected date of delivery March 11. Antibodies present throughout, titer not reduced by treatment with ethylene disulfonate. Section on January 28, 1948, because of pre-eclamptic toxemia. Tentative diagnosis hydrops fetalis. Baby died day before section. Baby very hydropic, wt. 2900 Gm., placenta weighed 2300 Gm. Autopsy, erythroblastosis, baby Rh positive, blocking antibodies in blood. Patient sterilized at section.

Case No. 8. Mrs. A. J. F.

Mother Rh negative, father Rh positive.

1st Pregnancy—1936, 7 lb. 13 oz., healthy.

2nd Pregnancy—1939, 9 lb. 10 oz., healthy.

Transfused nine times after this delivery because of septicemia. This was before discovery of Rh factor, some of the blood was probably Rh positive.

3rd Pregnancy—Delivered July 27, 1947, erythroblastotic child, Rh positive, lived twelve hours. Hydramnios present. Mother's blood seventeen days before delivery negative for Rh agglutinins, positive for moderately strong blocking antibody.

Case No. 9. Mrs. P. H.

Mother Rh negative, father Rh positive, homozygous.

1st Pregnancy—1943, 5¼ lb. healthy baby, Rh positive.

2nd Pregnancy—1947, delivery February 12. Mother had antibodies in last two months, titer increasing, prognosis for baby considered poor.

Delivered at term, 5 lb. 2½ oz., mild icterus, two transfusions, baby lived; Rh positive, no antibodies in baby's blood.

Case No. 10. Mrs. L. C. H.

1st Pregnancy—1939, term baby, normal.

2nd Pregnancy—1940, Cesarean section in Texas for placenta praevia, term baby, healthy.

3rd Pregnancy—1942, Section by E. L. K., term baby, healthy. No Rh test.

4th Pregnancy—1946. In fifth month found to be Rh negative, with some blocking and agglutinating antibodies. Husband Rh positive. Antibodies increased steadily. Section ten days ahead of estimated date of confinement. Baby hydropic, weighing 2925 gm. (placenta not weighed), lived

one hour. Autopsy—Erythroblastosis.

Case No. 11. Mrs. V.

1st Pregnancy—1930, premature 6 lb., living, Rh positive, jaundiced at birth.

2nd Pregnancy—1932, Miscarriage, 3½ months.

3rd Pregnancy—1934, premature, 7½ months, 4 lb., lived five days. Cause of death not known.

4th Pregnancy—1936, term, 5¾ lb., two transfusions from father, living, Rh negative.

5th Pregnancy—1941. Term—Intrauterine death two or three days before delivery. Autopsy?

6th Pregnancy—1942, slightly premature, 5¼ lb., no jaundice, living. Rh negative.

7th Pregnancy—Baby was delivered by section on August 13, 1946. Mother's serum showed no antibodies May 27, 1946, conglutination test positive 1:128, blocking test positive, 1:64. Baby was healthy and was found to be Rh negative. Hard to explain presence of antibodies in maternal blood. (Husband is Rh rh).

Another puzzler similar to Case 4.

Case No. 12. Mrs. E. W.

Mother Rh negative, father Rh positive.

1st Pregnancy—1939, healthy 7 lb. 9 oz.

2nd Pregnancy—1942, healthy, 7 lb. 10 oz.

3rd Pregnancy—Due about October 2, 1947.

Mother's blood August 22, 1947, strongly positive for Rh agglutinins and blocking antibody.

Section September 17, 1947, account titer of antibodies and no progress after fifteen hours of labor.

Baby hydropic, Rh positive, was transfused, did not survive.

CONCLUSIONS

1. All pregnant women should be tested for the Rh factor, if possible. This is especially important for the multigravida.

2. If the wife is Rh negative, the husband should be tested also. If he is negative, there will be no trouble.

3. If the wife is a multigravida, and the husband is positive, she should be tested for antibodies at monthly intervals from the fifth month on. If none are found the prognosis for the baby is good. If the antibody titer is low, and does not rise, the prognosis is still good. In case of a rising titer, the prognosis is not good.

4. Induction of labor or cesarean section two or three weeks before term is of little value.

5. If the husband is heterozygous, 50 per cent of his children will most probably be Rh negative, and hence are not in danger. If he is homozygous, and his wife has had one or more erythroblastotic babies, there is little chance of a healthy child in a subsequent pregnancy.

6. Babies clinically erythroblastotic should be transfused as often as indicated, using 20 to 30 cc. of compatible Rh negative blood each time.

7. Rh negative females requiring transfusion should receive only Rh negative blood.

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STREPTOMYCIN IN THE NEGRO WITH TUBERCULOSIS*

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Tuberculosis in the negro represents a relatively more serious and a more toxic disease than it does in the white race. The negro does not seem to have a natural resistance to tuberculosis. As a result, the type of cases seen in the negro hospital wards invariably represents a stage greater than minimal in almost 100 per cent of the cases. A substantial percentage are far-advanced cases, and many are complicated by tuberculosis laryngitis and pneumonitis.

From a curative outlook, our present therapeutic routines are, as often as not, of no avail in the negro with tuberculosis. Therefore, any procedure or drug that might act to stabilize, resolve, or cure the disease process, whether used alone or in conjunction with other procedures, is worthy of a full trial. Since streptomycin has been given sufficient clinical observation to indicate that it gives excellent

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clinical results, both palliatively and therapeutically, in certain types of tuberculous infections, its use is all the more indicated and needed in the negro with tuberculosis.

Unfortunately, this is easier said than done. Because of budget limitations, the hospital supply of the drug for tuberculous patients is allocated on an individual basis and final approval has been received for but very few patients, regardless of color. As a result, it has been necessary to have the patients purchase the drug themselves, whenever the staff has felt that the drug was indicated. Even though its usage has been severely limited by the staff, the economic status of most of the Negroes in the tuberculosis hospital wards precludes its use in almost 50 per cent of the cases. A dosage of 1 gm. per day of streptomycin[1, 2] has been shown to be effective in tuberculosis therapy, and at the same time to be less toxic and to produce less disturbance in the vestibular function.[3] Because of the economic factors involved, the smallest effective dosage of streptomycin is desirable in these negro patients. Therefore, the dosage finally determined for usage was 0.5 gm. twice a day, and in some instances of long periods of administration the dosage was decreased to 0.25 gm. twice a day for maintainance.

The use of the drug has been limited almost exclusively to the treatment of tuberculous pneumonia (both alone and as an adjunct therapeutic agent), as an adjunct with thoracoplasties, in the treatment and control of contralateral spreads and for tuberculous laryngitis. A total of 15 negroes have received streytomycin, some still receiving the drug, including 7 males and 8 females. The ages vary from 20 to 61.

The treatment of tuberculous pneumonias has been a great problem in the negro. Regardless of whether one uses bed rest, pneumothorax, phrenic crush or pneumoperitoneum, individually or in combination, the results have not proved consistently effective in stabilizing the pneumonic process. In almost all instances, the disease progresses rapidly to a fatal end. Four cases in this series were diagnosed as having tuberculous pneumonia and were given streptomycin as part of the therapeutic regime. There were 2 males and 2 females. One male had a lesion which was definitely stabilized, and probably improved, after two months of streptomycin in a dosage of 0.5 gm. twice a day. After this period, a thoracoplasty was started, but has not been completed, as of this date. The streptomycin is being continued until the surgery is completed, and at the present the lesions gives no evidence of extension or contralateral spread. The other 3 patients received pneumothorax as a specific therapy, and streptomycin was given as a supportive measure to prevent extension and contralateral spread. All were given 0.5 gm. twice a day prior to instituting pneumothorax. In all 3 instances, the pneumothorax was considered unsuccessful because the upper lobe was adhered and not amenable to pneumolysis. The 3 patients are now to be prepared for thoracoplasty and streptomycin will be continued as an adjunct to the surgery. Although the period of observation in each case has been relatively short, the drug has apparently prevented the usual contralateral spread, and fatal termination, in all 3 instances. As a rule, such a complication would have occurred by this date in the usual control case. The 4 cases include an age variance of from 25 to 47, so that age apparently plays no part in the results.

Seven cases of tuberculous laryngitis, three males and four females, were given streptomycin in a dosage of 0.5 gm. twice a day. The length of the treatments varied from eighteen to fifty-five days, averaging mostly thirty to forty days; the amount of streptomycin administered varying from 9 to 28 gms. and averaging about 15 to 20 gms. A review of the results shows that all 4 of the female cases received excellent symptomatic relief, and definite pathological improvement was

demonstrated by comparative studies, before and after the drug, as visualized by direct laryngoscopy. Of the males, only 1 received any definite symptomatic relief with confirmation by laryngoscopy, the other 2 showing only slight improvement clinically and by laryngoscopy.

Two females, age 30 and 32, are re-

CHART OF PATIENTS RECEIVING STREPTOMYCIN

Case	Age	Sex	Streptomycin		Diagnosis	Results and Comments
			Days	Gms.		
F. J.	32	F	90	90	Contralateral spread after thoracoplasty	No drug with thoracoplasty: large cavity completely closed, with a small residual infiltrate after 60 days of streptomycin
J. R.	35	M	42	42	Tuberculous laryngitis	Very slight symptomatic relief; complicated by TB endobronchitis
E. T.	25	F	55	55	Tuberculous laryngitis	Excellent symptomatic relief; complete resolution by laryngoscopy
E. B.	40	F	40	40	Tuberculous laryngitis	Marked symptomatic relief; received drug and then deserted
A. J.	53	M	36	36	Tuberculous laryngitis	No symptomatic relief; no improvement by laryngoscopy
V. S.	25	F	83*	139*	Tuberculous pneumonia	Stabilized by streptomycin; PNX unsuccessful; to have thoracoplasty
P. B.	42	M	64*	64*	Tuberculous pneumonia	Stabilized by streptomycin; thoracoplasty started, without spread or extension to date
V. L.	61	F	28	28	Tuberculous laryngitis	Marked symptomatic relief; complete resolution by laryngoscopy
J. F.	23	M	68*	64*	Large cavity on one side and infiltrate on other side	Infiltrate completely cleared and cavity much smaller after 60 days of streptomycin; prepared for a thoracoplasty as dosage decreased
A. B.	20	M	32*	26*	Unexpandable lung after PNX	No contralateral extension after 2 stages of thoracoplasty
M. A.	22	F	30	30	Tuberculous laryngitis	Marked symptomatic relief; marked improvement by laryngoscopy
W. C.	33	M	16	16	Tuberculous laryngitis	Excellent symptomatic relief; definite improvement by laryngoscopy
A. A.	47	M	45*	45*	Tuberculous pneumonia	Stabilized by streptomycin; thoracoplasty to be performed after unsuccessful pneumothoraces (due to broad adhesions)
E. C.	34	F	32*	32*	Tuberculous pneumonia	
R. T.	30	F	30*	30*	Contralateral spread after thoracoplasty	No drug with thoracoplasty; spread stabilized by streptomycin; PNX to be started and streptomycin continued

*Course of streptomycin not completed.

ceiving 1 gm. of streptomycin a day, in divided doses for the treatment of contralateral spreads which occurred subsequent to thoracoplasties. Neither had received streptomycin with the original thoracoplasty. In the first case a contralateral spread with a large apical cavity developed two years after the original thoracoplasty (Figure 1). After 60 days of

Figure 1. Contralateral apical cavity developing two years after thoracoplasty.

streptomycin, in a dosage of 0.5 gm. twice a day, the cavity closed completely and a small residual infiltrate remained (Figure 2). In the second case, a contralateral spread occurred six months after surgery. Thirty days of streptomycin in a similar dosage, resulted in stabilization of the contralateral lesion and probably some regression. The drug wil be continued for another sixty days.

As a therapeutic adjunct to thoracoplasty, 2 males were given streptomycin to prevent a contralateral spread. Past experience has shown this occurs during, or immediately after, most of the thoracoplasties performed in the negro. This seemed to result in spite of the fact that

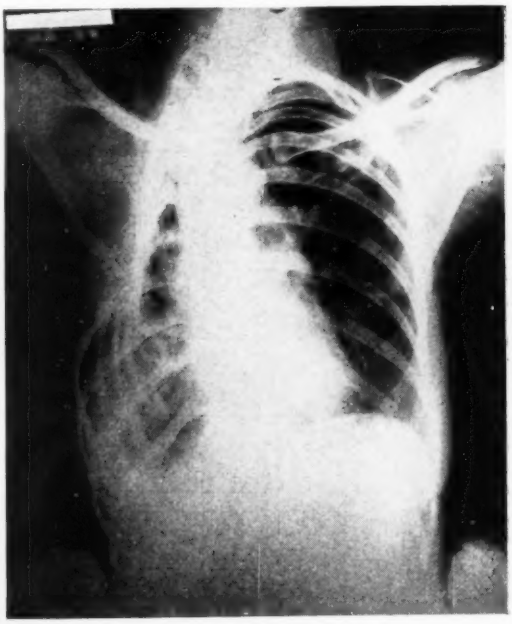

Figure 2. Same cavity closed after sixty days of streptomycin therapy.

extreme caution had been exercised in selecting cases for this type of surgical procedure. One patient received the drug for a month prior to the operation and has just completed the second stage of the surgery. The original dosage of 0.5 gm. has now been reduced to 0.25 gm. for maintainance. As of the present date, there is no indication of any contralateral extension. The other patient originally had a large cavity on one side, (which was the side for surgery), and a large area of infiltration on the other side (Figure 3). After sixty days of streptomycin in a dosage of 0.5 gm., twice a day, the area of infiltration has completely cleared and the original cavity is now much smaller. (Figure 4). Surgery will soon be started, and the streptomycin is being continued in a reduced dosage for an additional thirty days.

DISCUSSION AND SUMMARY

Because tuberculosis in the negro represents a far more serious problem than it does in the white race, an effort has been made to evaluate the results of streptomycin given to negro patients in a public

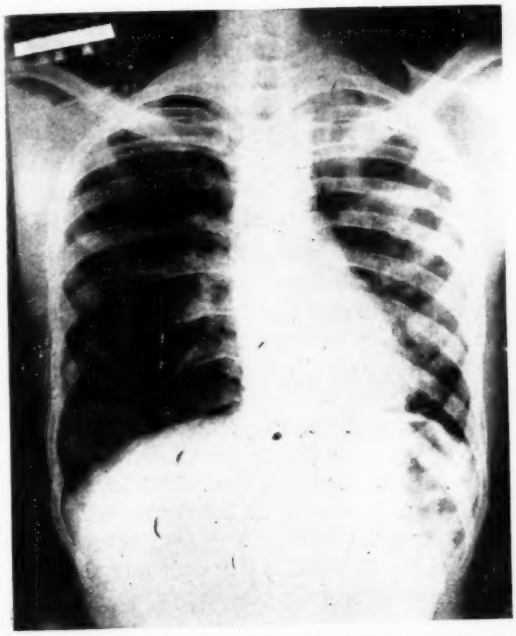

Figure 3. Large cavity and extensive infiltrate prior to streptomycin therapy.

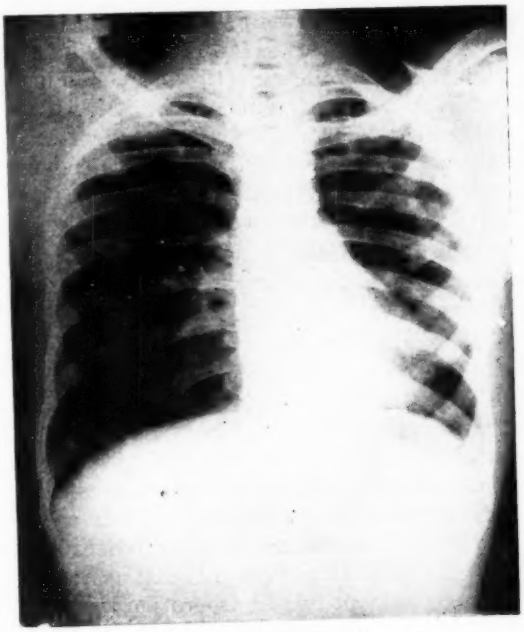

Figure 4. Infiltrate cleared and cavity smaller after sixty days of streptomycin therapy.

hospital ward. Fifteen negroes with tuberculosis were given streptomycin, as part of their therapy, in a dosage of 0.5 gm. twice a day initially, and in a dosage of 0.25 gm. twice a day, as a maintainance dosage in several instances.

An evaluation of the results shows that remarkable results have been obtained in stabilizing tuberculous pneumonias, when the drug is used as an adjunct with either thoracoplasty or pneumothorax. Similar excellent results have been obtained when the drug is used as a means of preventing contralateral spreads in patients undergoing thoracoplasties, and as a therapeutic agent in contralateral spreads occurring subsequent to thoracoplasties which were performed prior to the time streptomycin was available.

The results in tuberculous laryngitis have been encouraging but not spectacular, although one would have expected the best results in this type of patient. The fact that most of these patients were such far-advanced cases probably prevented more startling improvement in this condition.

Results seem to show no differentiation with regards to sex or age. Streptomycin was used only as an adjunct part of the therapeutic regime, and not as the sole therapeutic agent for the tuberculous infection.

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MORTON'S TOE

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Morton's toe is a chronic, disabling, clinical entity characterized by severe paroxysmal pain in the region of the fourth metatarsophalangeal joint. This syndrome is caused by tumefactive lesions involving the most lateral branch of the medial plantar nerve in the region of its bifurcation at the web between the third and fourth toes, and most probably results from the chronic trauma of ill-fitting shoes. Proliferative fibrosis of the nerve is a constant micro-

scopic feature. The exact histological classification of this lesion is too controversial to include in this presentation.

CLINICAL FEATURES

In the typical case, the patient complains of pain in the plantar aspect of the base of the fourth toe. The pain is severe, paroxysmal, usually radiates into the fourth toe, and occasionally the other toes. It may be burning, shooting, stabbing, or "like an electric shock." The attacks may last from several minutes to several hours in severe cases, are usually unaffected by any immediate therapy, and disappear spotaneously. Many patients in an effort to obtain relief, rest, remove the shoe, and massage the toe with occasional success. Walking at times is impossible. Numbness and tingling of the fourth toe may be present. At times the pains may radiate up the leg and thigh. With time the attacks increase in severity, frequency, and duration. Involvement is usually unilateral but not infrequently bilateral. In bilateral cases, the clinical onset in one foot is followed in the other by an interval of months to many years.

The most constant finding on examination is severe localized tenderness produced by digital compression deep in the web between the third and fourth toes. Quite often the radiating pain, if present, will be reproduced in this manner. Of extreme importance is the frequent presence of hypesthesia to pin-prick of the adjacent surfaces of the third and fourth toes. The tumor mass is rarely palpable. Occasionally pain is elicited on passive dorsiflexion of the third and fourth toes.

ANALYSIS OF CLINICAL MATERIAL

This presentation is based on a study of 12 patients who had surgical exploration of the lesion. Three patients had bilateral involvement. Five other patients with typical features refused operation and are not included.

All patients were white. Eight patients were female and four were male. Nine cases were unilateral and three bilateral. The left foot was affected in 8 instances and the right in 7. The ages varied from 24 to 66, with an average age of about 35 years. The duration of symptoms ranged from eight months to twenty years, with an average of about two years. In only 2 instances were the complaints atypical. Both patients were referred for sciatica, one bilateral and the other unilateral. It is interesting to note that in both cases, the direction of radiation was cephalad.

On physical examination, classical tenderness was elicited in all but one patient. In the unilateral cases, hypesthesia to pin-prick was noted in 6 instances and absent in 1. Two cases were not tested. In bilateral cases, it was present in both feet in one case, present in one foot and absent in one foot in one case, and absent in both feet in one case. Collectively, it was present in 9 of 13 feet tested. A tumor mass in the web between the third and fourth toes was palpated with certainty in only one patient. Deformities such as pes planus, and hallux valgus were noted in several cases. Bilateral plantar callosities beneath the metatarsal heads were present on only one occasion.

Radiographic examination of all feet included in this series failed to reveal any significant findings. (However, 2 other cases with typical clinical characteristics presented evidence of a degenerative arthritis localized to the fourth metatarsophalangeal joint. Unfortunately, exploration was not done in either case).

Routine laboratory examinations were essentially negative and all patients were seronegative.

Novocain infiltration of the web between the third and fourth toes was of value in differential diagnosis in doubtful cases. If symptoms were relieved immediately after infiltration, operation was indicated.

Conservative treatment was not considered in view of the pathological nature of the lesion. Furthermore, every patient had every conceivable type of non-operative therapy without relief. Operation was performed under local, spinal, or general anesthesia through either a dorsal longitudinal web incision or a transverse plantar approach distal to the heads of the metatarsals. The latter was used to explore the branches entering several webs. In every

case, the branch of the medial plantar nerve to the third and fourth toes was found to be enlarged in the region of its bifurcation. In some instances, it consisted only of fusiform thickening of the nerve proximal to its bifurcation. In others, a distinct nodular tumor mass involving the distal end of the nerve and its bifurcation was found. All lesions were excised and subjected to histological examination. These features will be presented in a subsequent publication. The largest lesion measured 1.5 by 1.0 by 0.4 cms. The smallest measured 0.8 by 0.4 by 0.3 cms. The size of the lesion bore no relationship to the duration and severity of symptoms.

All wounds healed *per primum* and the patients were ambulatory between the eighth and twelfth day. Postoperative numbness of the third and fourth toes rapidly disappeared except in one case. Ten patients, including three with bilateral involvement have been followed for over a year. Relief has been maintained in every case. Of the remaining two patients, operated within the past year, one patient still complains of pain in the operative scar on the dorsum of the foot.

ILLUSTRATIVE CASE REPORTS

Case 1. A 33 year old white female complained of pain in the plantar aspect of the base of the right fourth toe of eight months' duration. The pain became increasingly severe, radiated into the third and fourth toes and "burned something awful." At first relief could be obtained by changing into bedroom slippers. Later no treatment afforded any relief. On examination, severe tenderness was elicited in the web between the third and fourth toes of the right foot. A small tender mass the size of a pea was palpated deep in this web. Hypesthesia of the adjacent surfaces of the web between the third and fourth toes was present. X-ray and laboratory studies were negative. Operation through a dorsal web approach under local anesthesia revealed a nodular tumor involving the distal end of the most lateral branch of the medial plantar nerve and measuring 0.8 by 0.4 by 0.3 cms. Wound healing was uneventful. The patient was completely relieved of symptoms. She remains asymptomatic one year after operation.

Case 2. A 24 year old white female complained of severe pain in the left forefoot of one year's duration. The pain involved all the toes except the great toe and was associated with numbness of the toes. The attacks occurred daily and lasted several minutes to several hours. Partial relief was occasionally obtained by rubbing the toes. About a month before examination, similar symptoms developed in the right foot. Examination revealed tenderness in the web between the third and fourth toes of both feet. Hypesthesia was only present on the left. Bilateral tender plantar callosities were present beneath the metatarsal heads. Under pentothal anesthesia, both feet were explored through transverse plantar incisions exposing all of the webs. Typical fusiform lesions were found bilaterally between the third and fourth toes. Her postoperative course was uneventful and the patient was completely relieved of symptoms except for persistent numbness of the right fourth toe.

Case 3. A 32 year old white male was referred for sciatica. He complained of pain in the right foot radiating up the right leg and thigh. For several years he had suffered pain beneath the right fourth toe and had worn all types of metatarsal pads and shoe corrections without relief. During the past two weeks the pain became so severe, that he was forced to walk on the outer border of the foot. Examination was negative except for tenderness deep in the web between the third and fourth toes. No sensory changes were noted. Diagnostic novocain infiltration afforded complete relief for two hours. Exploration under local anesthesia through a dorsal web approach revealed a nodular tumor involving the nerve and wedged beneath the heads of the third and fourth metatarsals. Excision yielded complete relief of all symptoms.

SUMMARY

The characteristic features of Morton's toe, a painful clinical entity affecting the fourth toe, have been described. Surgical exploration in a series of 12 cases with involvement of 15 feet has consistently revealed a tumefactive lesion involving the most lateral branch of the medial plantar nerve in the region of its bifurcation at the web between the third and fourth toes. Excision of the lesion resulted in relief of preoperative symptoms in every case.

DIAGNOSIS AND TREATMENT OF COMMON VESICULAR LESIONS OF THE HANDS AND FEET

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We have selected this particular group of dermatoses as the subject of this paper because of its high incidence, the annoying incapacity that it occasions, and the high cost to the patient. Moreover, at this season of the year there is a sharp increase in the number of cases. Because patients so often seek relief by the use of home remedies obtained from drug stores, or well-meaning friends, they frequently develop long term eczemas which force them to accept lesser positions in their business or professional life, and to give up sports and hobbies.

This discussion will include only those lesions which exist primarily on the hands and feet, as the site of predilection, and not those which are simply part and parcel of an eruption elsewhere.

CLASSIFICATION:

1. *Hyperhidrosis*—Characterized by excessive sweating of the hands and feet, sometimes by the presence of fluid between epidermis and dermis.

2. *Pompholyx*, also called dyshidrosis, nervous or toxic rash. Characterized by symmetrical, deep-seated, sago seedlike vesicles along sides of fingers and toes.

3. *Fungus Infections*, vesicular type—Characterized by a picture that is strikingly similar to pompholyx, proved by the presence of fungus on scrapings or culture from lesions on the feet. Sometimes accompanied by "ids".

4. *Erosio Interdigitalis Blastomycetica*, yeast infection due to *Monilia albicans* plus soap and water — Characterized by wet, moist erosions between the fingers with elevated vesicular borders, paronychia and eponychia.

5. *Bactriids*—Characterized by a palmar and plantar pustular eruption. Lesions at first clear, becomes milky white and pustular, 1 to 5 mm. in size.

6. *Dermatitis Venenata* — Characterized by patchy, grouped, small superficial vesicles over dorsum of hands or fingers (as with rubber gloves) on dorsum of feet (due to shoes, nylons, etc.).

7. *Infectious Eczematoid Dermatitis* — These are the chronic recurrent eruptions that Unna, Engman, and more recently Lane, have written about. The original cause has been lost sight of and sensitization and infection have resulted.

HYPERHIDROSIS

Patients with hyperhidrosis are less of a problem in civilian life than they were in the Armed Forces. There are several reasons for this: First and foremost, the condition was greatly aggravated by heavy closed shoes, hot weather, and long fatiguing marches. This factor, plus the psychic trauma of combat warfare was enough to produce very aggravated cases of this condition. In extreme cases the entire plantar surfaces of the feet, together with an area halfway up the foot would be covered with bluish white, macerated skin, with fluid separating the epidermis and dermis. The fluid could be shifted about over a large area by palpation. The odor of the feet was terrific and the feet were cold and clammy to the touch. Severe cases were hospitalized and treated with 10 per cent formalin soaks. At first, severe cases were returned to the zone of the interior. Later on these were placed on limited duty overseas. A few were completely relieved by lumbar sympathectomy. These were the cases most subject to trench foot, and conversely, very severe hyperhidrosis

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was a frequent sequela of trench foot. In civilians, this is manifested by excessive sweating, cold, clammy, wet, bluish, odoriferous feet, and seldom by fluid separating the dermis and epidermis.

POMPHOLYX OR DYSHIDROSIS

Dyshidrosis was the term applied to this condition, because of the association of the disease with hyperhidrosis, by Tilbury Fox in 1837, who assumed incorrectly that the vesicles were the result of obliteration of the sweat ducts.* In pompholyx an eruption occurs on the sides of the fingers and toes, with scattered palmar and plantar patches. The vesicles that compose the eruption are deep-seated, thick-walled lesions, which are filled with a thick, clear, serous fluid, which becomes milky. Explosively recurrent outbreaks are usually typical for a given patient. Some present the disease only during times of stress and strain, others during hot, humid weather, others at each difficult menstrual period. Along with a great variety of causes most of which are associated with nervous exhaustion, much credence is given to a psychosomatic association in this condition. The symptoms are intense at times with burning, itching, and swelling of the fingers (and therefore stiffness). It is unaccompanied by epidermal allergy (patch tests are negative). The lesions regress rapidly in five to ten days, the vesicles which are superficial, rupture, and dry up and the deeper ones absorb in situ, leaving a brown discolored spot.

Treatment: Local therapy should consist only of cool, boric acid, or Burrows' solution (1-10) soaks for ten to fifteen minutes twice daily. Avoid the use of fungicides, ointments and greases in the acute stages of the disease. Lassars' zinc paste should be employed until the acute stage has subsided. Attention to the internal aspect of therapy in this condition will require more thought than local therapy. The psychosomatic aspect should be investigated and indicated therapy instituted. Mild sedatives, such as phenobarbital are of great value in this condition.

*Becker and Obermayer, Modern Dermatology and Syphilology. 2nd Ed. 1943.

FUNGUS INFECTIONS OF THE VESICULAR TYPE OF THE HANDS AND FEET

In our section of the country, the incidence of this type of eruption is relatively high. There is a sharp increase in the number of cases in hot, humid weather, especially if the hot humid weather is prolonged. This precipitates not only numbers of new cases, but greatly aggravates the current ones and causes yearly relapse. There are many difficulties to the differential diagnosis, especially from pompholyx. There are a few points of differentiation—the only sure way is the positive identification of the fungus on scraping or culture. Fungi are readily found in scrapings or culture from the lesions of the feet; however, it is unusual to find them in the lesions of the hands. These lesions are most commonly considered "ids". We use the same treatment as in the acute cases of pompholyx. After the acute phase, we have found that Desenex, Sopronol and Quinolor Compound Ointments used at night to affected areas of the feet are helpful.

DERMATOPHYTIDS

These are the scattered, grouped, pinpoint, superficial papulovesicular eruptions which are extremely pruritic and occur on the arms, upper trunk, face and neck. They are complications of an active fungus infection and are most apt to be present in patients with a vesicular type of fungus of the feet. This occurs especially in the acute stage after the injudicious use of fungicidal ointments.

Penicillin reactions cause an acute vesicular eruption. One of the more common types of penicillin reactions is manifested by a vesicular eruption not unlike that seen in pompholyx and fungus infections. This comes on during the first thirty-six to forty-eight hours following penicillin therapy. It is not a severe enough complication to warrant discontinuing the drug, and disappears within a few days after the medication is discontinued.

In many cases we find secondary infection by staphylococcus and streptococcus. In some of these we use penicillin. Because of the close relationship between fungi and penicillin, a violent reaction gives credence

Figure 1. Case No. 1, showing dermatophytids.

to the fact that in that particular case at least, the original cause of the condition was a fungus.

Case No. 1. Colored male, age 10, with an eruption on the arch of both feet, of eight days' duration. It began as deep blisters which appeared suddenly. He had received no therapy. Four days later he developed a rash on the arms, face, trunk and head, which itched severely.

Examination revealed deep-seated vesicles most of which were in the process of drying and others appeared as pigmented macules located on the arches of both feet. There were also grouped papulovesicular crusted lesions over the trunk, extremities and face.

Scrapings and cultures for fungus were positive from the feet.

BACTERIDS

This type of dermatosis is not often given a classification of its own, for there are many who believe that this is a subgroup of the infectious, eczematoid dermatitis. There are some of us, however, who believe that it is a true symptomatic condition, due to a focus of infection. The chief argument in favor of this stand is the temporary flare-up with ultimate cure when the focus is successfully removed. It presents a dermatosis composed of rather large vesicles which soon become pustular, on palmar and plantar surfaces. It is not common that we have a clear-cut case of this condition.

EROSIO INTERDIGITALIS BLASTOMYCETICA

This is an eruption which is found on the folds between the fingers. It often starts in the space between the ring and middle finger, and very often beneath the rings, because of soap accumulation. It is also an occupational disease for those who are exposed to soap and water, such as soda fountain workers, and bartenders. It starts as a raw area with small vesicles scattered usually on the periphery which soon rupture and give the margin a loose, sharply defined, undermined edge. Smears from these lesions reveal the typical budding spores.

Treatment: Strict avoidance of soap and

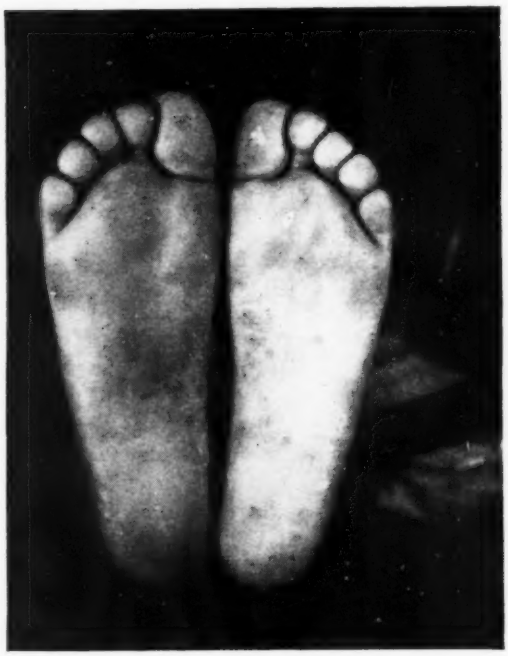

Figure 2. Case No. 1, showing vesicular lesions from which a positive scraping and culture for fungus were made.

water, mild soaks of cool potassium permanganate, boric acid, or Burrows' solution, followed by the application of 1 per cent aqueous solution of gentian violet.

DERMATITIS VENENATA (CONTACT DERMATITIS)

This is a patchy, sharply demarcated, erythematous, fine vesicular eruption, usually appearing on the dorsa of the hands and feet. The most common offending agents are leather, shoes, nylon stockings, rubber gloves, dye, and fungicidal drugs. Diagnosis is made from an accurate history, patch tests, or deliberate contact with the suspected substance. A negative patch test does not prove that the patient is not sensitive to the substance used, but a positive patch test is reasonably good evidence.

Case No. 2. White male, age 24 years, with a chronic, recurrent eruption, confined to the dorsal and medial aspects of both feet, of two years' duration. Previously treated for fungus infection on numerous occasions. He presented a sharply defined, erythematous and scaly eruption, at the sites mentioned above. Laboratory tests were negative except for positive patch test to the leather from his shoes. Deliberate exposure to his shoes produced recurrence of the lesions, after having been cleared.

Figure 3. Case No. 2. Dermatitis venenata due to leather.

Case No. 3. White male physician (general practioner) age 52, had a persistent eruption on the dorsa of both hands for the past eight years with a definite contact history associated with wearing rubber gloves. The eruption consisted of an erythematous, papulovesicular and scaly weeping eczema limited to the dorsa of both hands and fingers. Clinical cure was obtained with the use of Neoprene gloves to which he was patch test negative.

INFECTIOUS ECZEMATOID DERMATITIS

This is a group of vesicular dermatoses in which we, as dermatologists, are deeply interested. It is a chronic, recurrent, vesicular eruption of the hands and feet that seldom presents the same history or clinical picture. These dermatoses in origin are probably pompholyx, dermatitis venenata, fungus, or any one of the aforementioned conditions and others. Many now believe in the original theory of Unna that the condition is one of infection. It is our plan for the future to carry on further studies in an attempt to learn more about the condition in order to stabilize our therapy.

The clinical picture as quoted from Lane *et al** is similar to that which we encounter. "There has been a great variation in the type, arrangement, distribution, and extent of the lesions seen in these patients; some show a minimum of involvement with a few scattered vesicles on the lateral aspects of one or more fingers, others show a generalized papular vesicular eruption, covering almost the entire area of both hands and at the same time extending to the forearms. Most frequent sites of beginning of the eruption seem to be lateral and dorsal aspects of the fingers, uni or bilateral. It usually starts as a patchy eruption which may or may not become confluent or diffuse. One of the first symptoms is pruritis. There is usually peripheral extension of the patches, with a tendency toward a concentration of the vesicles at the edges of the lesions. Rupture of vesicles results in a serous exudate and formation of crusts. Pustules are frequently seen. Palmar vesicles are deeper and have undermined edges."

Case No. 4. White female, married, age 34. History of eruption beneath the wedding ring, recurrent each summer for the past six years. This summer, patient presented a rash of four days' duration, which was of sudden onset and involved

———*Lane, C. G., Rockwood, E. M., Sawyer, C. S. and Blank, I. H., Dermatoses of the Hands. J. A. M. A. 128:987-993, Aug. 4, 1945.

the hands and arms which were greatly swollen and covered with vesicles, ranging from 1 to 5 cm. in diameter. Scrapings and cultures for fungi were negative. She was placed on sulfadiazine, gms. 1, q.i.d., Burrows' soaks 1-10 for 10 min. b.i.d. and instructed to avoid all chemicals and soaps. She returned three days later greatly improved. Because some of the lesions were pustular, penicillin was added to the treatment, 50,000 units every three hours. She returned two days later with the entire process greatly flared up. The penicillin was discontinued but the sulfadiazine and soaks were continued. On her return three days later, the condition had again subsided and she cleared up entirely on this routine in ten days.

Was the original condition due to erosio interdigitalis blastomycetica and thus the flare-up indicating a fungus as the etiological agent?

Figue 4. Case No. 5. Infectious eczematoid dermatitis.

Case No. 5. White male 27 years old with history of intermittent vesicopustular eruption of the hands for two years. He had been treated with local measures in the hospital on two previous occasions. Examination revealed vesicles and pustules in the palms and on the sides of the fingers of both hands, with considerable thick crust formation. Scrapings and cultures for fungi were negative, search for foci of infection negative. Patient was placed on boric acid compresses, the lesions were debrided and in addition he received x-ray treatment to the hands. Under this regime the skin at the sites of involvement became free of any vesicles or pustules.

SUMMARY

At present our approach in the study and care of these cases is as follows:

I. History

a. Past treatment history — (1) Drugs (2) Reactions (3) X-ray treatments

b. Seasonal incidence

c. Exposure to fungus

d. Occupation and hobbies — (1) Chemical and plant contacts including soaps.

II. Physical examination and search for foci of infection.

III. Character and distribution of lesions.

IV. Laboratory aids.

a. Scrapings and cultures

b. Skin tests with Trichonphytin and oidiomycin

c. Patch tests with suspected contactants.

V. Therapeutic response.

The therapy is dependent upon the results of the above investigations. For those specific entities previously discussed we have outlined respective treatment. For this last group which is called infectious eczematoid dermatitis our therapy is as follows:

I. General.

a. Improvement of general health

b. Correction of predisposing factors.

II. Internal Medications.

a. Sulfonamides

b. Penicillin.

III. Local therapy.

a. Compresses, cool—(1) Burrows' Solution. 1-10. (2) Potassium permanganate 1-6,000. (3) Boric acid solution 2 per cent.

b. Topical applications — (1) Lassars' Paste USP.

IV. Miscellaneous factors.

a. Avoid soaps, chemicals, irritants, rubber gloves and immersion of hands in water.

CONCLUSIONS

The more common vesicular lesions of the hands and feet have been briefly discussed, and we have outlined our method of diagnosis and treatment of each condition. If not carefully handled, any one of

them may become a chronic affair, and fall into the group of infectious eczematoid dermatitis.

DISCUSSION

Dr. J. K. Howles (New Orleans): I think Dr. Kennedy should be congratulated on bringing to our attention the most common dermatological condition encountered in this vicinity.

There are a few points I would like to stress: One of my assistants, who had recently started training, mentioned to me that he noticed that rarely do you find a true vesicular eruption of the hands, uncomplicated. On thinking of it, we rarely see uncomplicated vesicular eruptions of the hands. Most of the contact dermatitis we encounter in housewives and other people are occupational dermatoses acquired in their work, principally due to too strong soaps, grease solvents, etc., used to clean their hands.

One point mentioned was that of avoiding rubber gloves. Rubber gloves seem to contain some substance today that irritates. It is not as irritating when one substitutes non-allergic rubber gloves. A simple method, when using rubber gloves in household work is to put on a thin cotton or linen glove under the rubber ones. This absorbs perspiration containing irritating substances which produce reaction on the skin.

Dr. Kennedy mentioned the necessity of opening the vesicles when they occur, in order to cut down secondary infection. No one dies of superficial fungus infection but persons are incapacitated and the morbidity is enhanced by failing to cut down secondary infection by opening vesicles. The areas are going to peel anyway.

Dr. Kennedy referred to fungicidal agents. I am sure he means fungistatic. Phenol is a fungicidal and also a protein digestant. Most fungicidal agents are protein digestants.

Dr. Medd Henington (New Orleans): There was one thing that came to mind when Dr. Kennedy was presenting this paper. That is, I firmly believe that too active a treatment for an acute fungus infection of the feet is in many instances responsible for the "ids" that occur elsewhere on the body, particularly the hands. This has been demonstrated so many times that recently it has been my policy to treat these acute vesicular eruptions of the feet with boric acid compresses and talcum powder during the acute stage. When the condition has subsided somewhat, then we go ahead with the fungistatic medications. We have also found that a small amount of x-ray to the involved parts will reduce the vesicular formation and relieve the itching all of which is gratefully received by the patient.

PREOPERATIVE CARE OF PATIENTS FOR INTRAOCULAR SURGERY*

WALTER STEVENSON, M. D.

QUINCY, ILL.

While the title of this presentation indicates that a discussion of the preoperative care of patients for all types of ocular surgery will be given, I feel that if my remarks are confined to the patient for cataract surgery they will cover fairly well all types of ocular surgery.

This premise involves a great many factors; among these are the type of patient, the age of the patient, the type of family, the associates of the patient, the degree of loss of vision, the general state of health, the refraction, and perhaps many other considerations.

TYPE OF PATIENT

Explanations that are required vary according to the patient. Some patients may be told that they have lens opacities which will probably become surgical cataracts even when vision is reduced only to 20-25. A stolid sensible individual will be helped by a plain statement of the situation. Such a one is apt to have noticed blurring of vision, that lights, especially at night, disturb him and probably will observe that despite many refractions here, there and everywhere, dissatisfaction with his degree of vision still remains. He should be told that no one can furnish glasses to his entire satisfaction because during the course of the attrition of age everyone develops a certain degree of lens swelling which causes frequent changes in refraction; that in his particular case this has progressed to the point where frequent changes of refraction are apt to take place; that in all probability frequent changes in glasses will be necessary. The present state of vision, or approximately such vision, may be maintained for many years by frequent changes in the strength of his glasses. Furthermore, that useful vision is not necessarily 20-20 or 20-15 vision and that even 20-40 vision is

*Presented before the Louisiana-Mississippi Ophthalmological and Otolaryngological Society, New Orleans, Louisiana, April 17, 1948.

useful vision. This type of patient will then be satisfied with such advice and return frequently for observation and maintain confidence in his doctor. Finally, this type of patient can eventually be informed if cataract surgery is becoming imminent and he will then accept the decision graciously and gratefully. However, the apprehensive introspective type of patient with a doting family, one who has been frightened by friends, and one who has a fear of cataracts should not be so informed, but a responsible member of the family should be advised of the real diagnosis.

A different problem is presented by the case of a Federal judge who was my golf partner for many years. He had lens changes three years before surgery and constantly complanied to me, especially on the golf course, that he would certainly have to go to some other ophthalmologist because I did not seem to be able to furnish glasses to his entire satisfaction. While a part of this attitude was facetious, still he was seriously concerned. This man was a highly apprehensive individual and I knew from the type of cataract he was developing that there would be slow progress. I did not want to throw him into a "tailspin" by telling him the truth about his condition. However, I did tell his wife and in that way controlled the situation. At the proper time I told him of his trouble, told him I had not informed him before and why. When this man came to surgery he was an excellent patient with full confidence in my judgment.

I have another type of patient, that of a wealthy widow who lives with an old maid sister, both the recluse type. This patient, even now, is almost ready for surgery but I have not told her of the seriousness of her condition and she seems well satisfied because the sister has been well informed and is able to control the situation when the patient complains of the doctor who does not seem to improve her failing vision.

I quote from a personal communication from Dr. Meyer Wiener, with whom I was associated many years ago, regarding his attitude about this important feature of advice to prospective cataract patients.

"There is danger in withholding information about his condition, even if the next of kin is notified. He never has the same confidence in the doctor, feeling that perhaps some information is kept from him. I have told my patients who were nervous and apprehensive that they have an opacity in their lens. That everybody past 60 has opacities; that opacities in the lens are called cataract, but that they seldom advance enough to seriously interfere with useful vision, and if they do, that operation will give them useful vision and that if I had anything interfering with my sight, I would rather have cataract than anything I know, because it is more amenable to treatment. Otherwise, I consider your advice sound."

AGE

Certainly it is useless to tell a person of 75 or 80 with 20-40 vision that he may need surgery, particularly if the cataract is progressing slowly. In all probability death will solve all visual problems for this type of patient and, as stated before, so long as 20-40 vision is obtained, surely sufficient vision is present; so why burden such an individual with additional worries?

OCCUPATION

One sould approach this phase with due consideration. A man of 50 whose livelihood depends upon good vision, and especially one who drives here and there as a business duty, must have better vision than a farmer who may quite easily continue his work with 20-60 vision. This type of patient should be told at once that he is losing vision and that when he is uncomfortable and cannot follow his vocation that surgery is available. Likewise, a housewife can do her work with greater reduction of vision than a woman who is employed in the business world. Obviously, a patient in poor health with vision impaired by lens opacities should not be burdened by a disclosure that cataracts are apt to further disable his vision. Frequent changes of glasses will probably satisfy the patient and some member of the family can be informed that the care of the general health is of greater importance than any local treatment.

INDIVIDUAL

The individual patient's reaction to his visual loss is certainly a factor in preoperative care. Whether or not a patient with cataract is to be operated upon, will depend

upon the patient's reaction to his own visual loss. Many patients complain bitterly of vision of 20-40 or 20-50, while others with similar visual requirements for their daily activities are happy with 20-80 or 20-100.

REFRACTION

In so far as refraction is concerned, innumerable patients with reduced vision come for observation with less vision than on a previous visit, and yet, for many years visual acuity can be restored to approximately its former state by minor changes in the lenses prescribed.

GENERAL HEALTH

The first step in caring for a preoperative cataract patient is to insist on a complete, general physical examination by an expert internist. This is sometimes a difficult problem, for many patients will insist that the examination be made by the family physician who is unqualified to make such examinations, despite the fact that the patient has implicit confidence in him. Therefore, it is unwise and hazardous to select any but competent physicians for such examinations even at the risk of offending the family physician.

Whether or not correction of physical defects inhibits the progress of cataracts, such faults must be corrected and it is my firm belief that in many instances cataract progress has been delayed by correction of physical defects, the administration of tonic medicines, and a change in the health habits of the individual.

Diabetes is not a contraindication. All cataract patients with diabetes should have a careful survey made by a competent internist, and when and if such a patient is stabilized, surgery is almost as safe in this type of individual as in an otherwise normal patient.

Hypertensive cases offer considerable risk to successful surgery. However, with a period of rest in bed, together with appropriate treatment, these cases can in almost every instance be successfully operated upon. The same statement may be made regarding cases of nephritis with high retention of non-protein nitrogen. Under hospital regime, strict diet, etc., these cases may be controlled. As to the highly neurotic, apprehensive patient, one may quiet his fears by a period of bed rest and judicious use of sedatives. I recall well when I was with Dr. Wiener that he did not consider hypertension a contraindication to intraocular surgery. Again quoting from his recent communication to me regarding this:

> "I don't agree with you about the risk in hypertensive cases. I have never had what I thought to be a serious complication due to hypertension. That is a matter of personal equation, however."

Of course a preoperative requisite is careful observation of the intraocular tension, both before and after dilating the pupil. Slit lamp examination of course is a prerequisite. Needless to say one should be extremely painstaking in his search for infections about the eyelashes, lids and conjunctiva. It is a very good practice to irrigate the lacrimal sac with a mild antiseptic before sending the patient to the hospital. It has been said that a dental examination and roentgenogram of all of the teeth should be a preoperative routine. While I am not opposed to this, it many times is not an expedient thing to do. It has been my observation through many years, and particularly in India, that this is not necessary. However, if it is done and any teeth are extracted, surgery should be postponed for at least two months.

INDICATIONS FOR SURGERY

When should a patient be urged to have cataract surgery? A wise surgeon will suggest to a cataract patient that he will assume responsibility for surgery when and if the patient requests such surgery. Don't ever urgently advise surgery unless the type of cataract suggests impending glaucoma, provided the patient has reasonably useful vision in the other eye. If, however, the cataract is mature or hypermature, I suggest surgery in such an eye regardless of the state of vision in the other eye. In other words, I leave it pretty much to the patient as to when he would like to have better vision. I usually state that when they are unhappy with their present vision, perhaps are becoming despondent and feel that they can no longer carry on the type of

life they desire, that then is the time to operate. Woe to the surgeon who operates upon an eye very defective in vision with a fellow eye in which fairly good vision is present, say up to 20-50. No amount of explanation before surgery will satisfy that patient when, after complete recovery of the operated eye, he asks, "Doctor, when do I get my glasses?" Despite every possible method to convince him of the fact that he was told that glasses could not be fitted until the fellow eye became more defective the patient does not understand "Why." So, if possible, wait until the fellow eye is so defective in vision that diplopia will not result with glasses.

There are definite exceptions to this. I have in mind a patient who came to me when he was the superintendent of signals for a large railroad system. He was almost totally blind in one eye with 20-20 vision in the other. On one of his annual examinations by the railroad service he was told that he could not remain on duty unless vision was improved in the blind eye. I told this man that he would not be able to wear a cataract lens with the vision as good as he had in the other eye. He said that all he wanted was to be able to sit down with a cataract lens on and pass the examination. This was accomplished by surgery and to my great surprise he appeared in my office sometime later wearing a cataract lens on one eye with a very minor strength lens in the other, stating that he had no difficulty whatever maintaining binocular vision and never had diplopia. I have had this happen a number of times—all of which proves that there are exceptions to everything.

So far as I know there is no definite method of determining the condition of the macula in the eye with a lens so opaque that the fundus picture is obscure. However, if the patient has good light perception, good light projection, and good pupillary reaction to light, together with the ability to recognize red, blue, and green colors through a pin hole, one can feel reasonably sure that the macula is healthy. Dr. Wiener has stated that flashing a strong light in the macular region of the cataract eye will produce a black central spot as an after-image, if the macula is healthy.

PREOPERATIVE HOSPITAL CARE

The patient should be placed in a hospital at least two days before surgery. A complete physical examination should be done again and, while bed rest is not essential before surgery, the hospital environment will be accepted and he will become accustomed to his surroundings, and calmness, so essential to an acquiescent patient, will be established. It is always wise to inquire if the patient has some particular medicine that he is in the habit of taking. Unless seriously contraindicated, by all means let him bring it along and take it. This applies especially to laxatives. Always inquire what laxative a patient is in the habit of using when such medicine is necessary and no matter what type of laxative that may be, that is the laxative that the patient receives. It is our practice to have a culture made of the conjunctival sac secretions and if pyogenic organisms are found surgery is deferred. Yet, through the years of my surgical experience I am convinced that patching an eye is sufficient evidence of cleanliness if a large amount of secretion does not accumulate beneath the patch after twenty-four to thirty-six hours. I do not use conjunctival antiseptics prior to surgery, as a rule, nor do I irrigate the conjunctival sac immediately before surgery. It is my opinion that protective lysozymes in the conjunctival secretion are best left undisturbed.

It is our practice to give each patient 150,000 units of penicillin in wax and oil two hours before surgery, and 150,000 units six hours following surgery. This is accepted prophylactic practice. Twenty-four hours prior to surgery ½ grain of phenobarbital is given orally three times daily. The face is washed with soap and water three times a day during this period, the hair having been shampooed before entrance into the hospital. A saline enema is given the afternoon before surgery—not the night before or on the day of surgery. At bedtime the night before, 1½ grains of nembutal or seconal is given by

mouth. An hour before surgery this same dose is repeated. An order prohibiting relatives or visitors in the room before surgery is imperative. My hospital has strict instructions not to disturb the patient until the moment of transportation to the operating room and as little excitement and noise as possible should be assured. Upon entrance to the operating room, noise of pans, conversation, etc., is forbidden and no one is permitted to initiate conversation except the surgeon and no one permitted to give instructions to the patient except the chief actor—the surgeon.

Placed upon the operating table, the patient is again assured that he is a good prospect for surgery, that a successful result is to be anticipated, and that with the exception of a slight pin prick for the initiation of anesthesia, that no pain will be experienced. The patient should be advised why general anesthesia is not practical. The day before surgery the eyebrow is completely shaved and the tips of the eyelashes are clipped off with the scissors dipped in vaseline. It is well not to clip the eyelashes too short.

ANESTHESIA

Despite the fact that the incision for cataract surgery is considered the most important factor to a safe and easy delivery of the lens, I personally consider good anesthesia equally important, if not more so. Whether one prefers to use cocaine or pontocaine as a topical anesthetic is a matter of choice. However, whichever is chosen it should be used in the patient's room before the journey into surgery. We use two drops of 5 per cent cocaine hydrochloride solution, five minutes apart, ten minutes before the patient leaves the room. After arrival upon the operating table, 4 additional drops of cocaine are distilled into the eye to be operated upon and 1 drop into the opposite eye. It is well to mention here, as a minor detail, that in dropping an anesthetic into an eye, or as a matter of fact, before dropping any liquid into an eye, one should consider that the closer the dropper is held to the eye the less shock of impact is felt by the patient. I consider this a very important detail. Remember that in most instances the patient is blind and cannot see what is going on and for that reason cannot anticipate what is to happen. Therefore, the surgeon should tell the patient, in so far as practical, everything that he is about to do before he does it. During this time akinesia is induced according to the method of Cecil O'Brien. This method has always proved entirely satisfactory to me, but there is a trick about its induction. The injection should be made directly forward from the incisura of the ear and about one-half inch from it. If one starts the injection immediately upon entry of the needle and keeps the procaine flowing ahead of the needle, this can be done practically painlessly. The procaine should be 1 per cent with 1 to 100,000 epinephrine chloride. The needle should be forced directly inward until the condyle of the jaw is felt. If the patient is edentulous the mouth should be slightly open in order to place the condyle of the jaw in the proper position. Having felt the bone with the needle point, the needle is slightly withdrawn and at least 4 cc. of procaine injected at this spot. If one keeps the needle in constant contact with the periosteum of the bone a considerable degree of pain will be induced. Therefore, the needle should be slightly withdrawn before injection. Upon withdrawal, gentle massage of the area should be made for three or four minutes. If this injection is to be effective, one will notice in thirty to forty seconds after injection that, while the eyes are closed initially, the lid on the injected side will open about one eighth of an inch and gradually open quite a little more. If this does not take place as stated a second injection should be made at the same point by a needle fanned out in three directions, up and forward, down and forward, and straightforward, injecting an additional 2 cc. of procaine. Following this the patient is directed to look down and 2 drops of procaine are injected just behind the tendon of the superior rectus muscle. Two drops of the same solution are then injected into the skin of the nose just above the inner canthus. Four or five drops are now injected into and below the outer canthus in

the event that canthotomy seems desirable. Through the lower lid below the tarsus and fairly close to the outer canthus 2 cc. of the same solution is now injected behind the globe. The needle should be directed to a point half way between the optic foramen and the posterior surface of the eyeball. To avoid hemorrhage one should adopt the method of injection devised by Harvey Cushing and this is to keep a slight amount of solution flowing from the end of the needle in order to push blood vessels aside. There will be a moderate degree of resistance to this until one feels the needle fall into the muscle cone space. Then 2 cc. of the solution should be injected. A word of warning regarding retrobulbar injection: It should not be done too long before the surgery because the eye may become extremely soft. While I was in India I saw a lens lost into the vitreous on three occasions because of a soft eye made so by retrobulbar injection given too soon. Tincture of merthiolate is now painted over the entire half of the face on the operative side, extending one-half to the opposite side of the nose and on the forehead at least as far across as above the other eye. In painting the lids one should pull the lids to the outer canthus with a piece of gauze in order to tighten the palpebral fissure so as to avoid merthiolate seeping through the fissure. Merthiolate in the conjunctival sac will congest to the eye to the extent that serious bleeding may ensue. Following the instillation of several drops of epinephrine chloride (1 to 1,000) the patient should now be ready for surgery after being draped. We drape the patient as follows: A large towel folded triangularly covers the hair and is held together with a towel clip. A large table cover is placed beneath the head and with the patient's head held above this sheet the eye-mask is placed over the operative eye and this mask is tucked well beneath the patient's head. This latter maneuver avoids displacement of the mask quite thoroughly. Sterile sheets are then draped over the patient's entire body, and the patient is now ready for the surgeon.

PRIMARY GLAUCOMA
NEWER TRENDS IN TREATMENT

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Glaucoma ranks third in the causes of blindness to those past the age of forty. Its frequence is about that of diabetes, approximating .1 per cent of the total population and 1 per cent in those past forty. The glaucomatous in this country number between 100,000 and 150,000. Those of the world number approximately 1,000,000 which is almost twice the population of New Orleans.

After more than one hundred years of investigation, primary glaucoma especially still remains ophthalmology's Public Enemy No. 1. Basically, the reasons are simple.

A. Glaucoma has been looked upon in the past as a single one dimensional entity such as a linear inch, with only one cause which was expected to explain all stages of all cases completely. Now we are beginning to realize that no one single cause is or can be completely explanatory in any and all stages of all cases of primary or secondary glaucoma. The causation of glaucoma, like that of other disease processes, is becoming increasingly recognized as being essentially a three dimensional problem, such as a cubic inch. All three dimensions or components exist in all cases, but their relative importance varies greatly in individual cases. These basic components are: Constitution, environment, and time. By constitution is meant the sum or total of hereditary characteristics, the potential basic structural material and functional activity of each cell or/and cell group from conception to death. Environment includes the normal and abnormal physical and chemical influences to which all cells are exposed during their lives. Time includes the life cycle or duration of all body cells both as individuals or groups, in the different stages of life; also their de-

The author is indebted to Smith, Kline and French and to Merck & Co. for the Furmethide and D.F.P. used.

struction and replacement rate. This three dimensional concept of causation in medicine may seem new, but two thousand years ago it was old. You have probably recognized the parable of the sower and the seed from the New Testament which crystallizes a basic law of all life which medical science has neglected.

B. The causation of glaucoma also largely remained a mystery until the physical and chemical fundamentals of the fluid in-go and out-go of the eye were reasonably understood. Glaucoma is essentially an imbalance between the in-go and out-go of intra-ocular fluids. The airplane was not practically possible until lightweight, efficient power and fuel were available. Similarly, glaucoma advancement in prevention and treatment was not practically possible until intra-ocular mechanics were reasonably understood.

Before looking into the future of glaucoma, a simple prevue of our present conceptions is desirable, especially to non-ophthalmologist physicians. The following concept of primary glaucoma is largely based on the writings of three world authorities on this subject. They are the late M. Schoenberg of New York, A. Busacca of Brazil, and A. Magitot of France. Primary glaucoma is essentially a bilateral progressive constitutional degenerative ocular disease, generally characterized by increased intra-ocular tension, peripheral and central visual deterioration, disc excavation and variable vascular reactions. In varying degrees it involves both the intra-ocular filtering mechanism and the fluid which must pass through that filter to exit from the eyeball. The filtering mechanism is located principally in the cornealscleral junction. The filtering fluid is quantitatively and qualitatively regulated by the retinal and especially the choroidal capillaries and precapillaries which in the glaucomatous are abnormal. This intra-ocular vascular degeneration is only a local part of a general bodily vascular degeneration. Capillaroscopic tests of the subungual capillaries show specific changes in the glaucomatous. These same vascular changes especially in the thalamic portion of the brain are vitally important. Here sensory impressions from the eyes and the other senses are transformed into the several types of autonomic impulses, including vasomotor. In the glaucomatous, emotional disturbances produce abnormal vasomotor reactions. These centrally and peripherally adversely affect the filterability of the intra-ocular fluids. It has long been known that fear, worry, anger, and anxiety of any form usually aggravate or precipitate glaucoma.

This brings us to the first major advance in the treatment of glaucoma: its hygienic care, prevention or retardation. Irrespective of other treatment, all glaucoma patients should be made to understand clearly and promptly that they must think calmly, act calmly and be calm to potentially retain their sight. Living dangerously to them frequently means dying blind. This hygienic aid to other treatment must obviously be used constructively and practically. It is neither time consuming nor complicated. During approximately five years in which I have successfully used it with hundreds of patients, I have not infrequently marvelled at the success with which the average glaucoma patient can overcome psychologic difficulties that seem almost unsurmountable. The glaucomatous must also learn to practice order and moderation in their daily physical and mental living habits. Moderation in the use of stimulants, especially caffeine, is important.

Before discussing the therapeutic treatments of tomorrow, let us review those of today. Pilocarpine suitably administered increases sweating; some of the bodily secretions become quantitatively increased and are also made thinner and more filterable. Atropine causes dryness of the throat, thus decreasing oral secretion quantitatively and making it thicker and less filterable. The former stimulates the parasympathetic mechanism; the latter paralyzes it. Generally speaking, atropine in the predisposed, therefore, precipitates or aggravates glaucoma while pilocarpine in-

creases intra-ocular filterability and is the most frequently used drug in the treatment of glaucoma. The technicalities of the intra-ocular mimetic mechanism are of so little interest to nonophthalmologists that they are omitted.

Two of the newer and therefore more experimental drugs apparently have the greatest future possibilities. The first is Furmethide, a 10 per cent solution of furfurylmethlammonium iodide (Smith, Kline and French), which clinically resembles pilocarpine, but is more powerful. In higher intra-ocular tensions, that is 35 mm. of mercury or more, it is instilled at fifteen minute intervals for two hours, after which it is instilled three to five times daily. In lower tensions, Furmethide is instilled three to five times daily with or without auxiliary drugs. During more than six months we have used Furmethide in approximately 20 patients whose glaucomas were not reasonably controlled by other miotics and who were poor surgical risks. It has been almost uniformly successful in reducing tension and or reasonably retarding progress of the disease. In one case tension was very slightly reduced. In 3 other cases the disease slightly advanced during six months, but much less than it had with other treatment. Furmethide is apparently best reserved for patients in whom other miotics are partially successful and as an emergency aid in acute glaucomas. It is not irritating and may be used with other miotics, especially D.F.P.

The second experimental drug of apparent future value is D.F.P. (diosopropyl flurophosphate, Merck). This powerful miotic of the eserine group is five times stronger than a .02 solution of eserine. Being unstable in aqueous solution it is manufactured as a 1 10 per cent solution in peanut oil. Although locally nonirritating, it should be used as infrequently as practical, ranging from twice daily to twice weekly. Blurred sight from spastic accommodation and transient myopia as well as occasional headaches follow its use. In approximately 20 cases in which we have used this drug, tension was at first usually promptly reduced; but its tension reducing qualities in at least 4 cases diminished materially during the first three months. It was then discontinued or supplemented with Furmethide, pilocarpine, or other auxiliary drugs. Unfortunately, only clinical tests are as yet available to determine the specific miotic best adapted to any given case.

All primary glaucoma operations are designed to increase the filtration area and or reduce the aqueous. There have been few or no basic advances in glaucoma surgery during the past decade. Goniotomy is a streamlined de Vincentii operation. When therapeutic and hygienic treatment will apparently not afford any glaucoma patient reasonable sight during life, the surgical risks involved should be considered promptly. Glaucoma progress is measured in terms of peripheral and central vision as well as ocular tension. The success or failure of any glaucoma operation is explained in four questions:

1. Did the operation quantitatively and qualitatively meet the specific needs of the affected eye?
2. Were the risks justified?
3. Was the operation correctly performed?
4. Did the eye survive the operative trauma?

Let us not forget that more than 60 per cent of all glaucoma operations are not successful in normalizing tension and maintaining central and peripheral vision for three years. We should also remember that glaucoma operations do not cure glaucoma. They may normalize tension, but they do not normalize the abnormal quality of the intra-ocular fluid. Among promising technics of tomorrow are anterior chamber air injections and goniotomy. The former is used at the termination of practically all glaucoma operations to facilitate filtration. The latter is apparently of most value in specific types of infantile glaucoma.

The anterior chamber angle varies more widely in the glaucomatous than in normals and is accurately studied with the gonioscope. The relationships in any specific

case between the depth of the anterior chamber angle and the types of glaucoma (vascular or corneoscleral factor dominance) as well as the most effective type of medical or surgical treatment are not yet sufficiently understood to be of great practical value. Generally speaking, in the narrow angle groups with lower tensions the trend is toward goniotomy and cyclodialysis; and in higher tensions, toward iris inclusion operations. In the wide angle group, the same operations are used in lower tensions, and corneosclerectomy in higher tensions. If transient edema is the primary emergency, as in acute glaucomas, sclero-iridectomy is the operation of choice. Obviously, tension should be normalized therapeutically as far as possible before surgery is attempted.

Newer trends in treatment really mean the probable remedies of tomorrow. In their discussion the fact is easily overlooked that the great progress which has been made in the sight conservation of the glaucomatous, especially in the past decade, has not been due to any single, spectacular or epoch-making medical or/and surgical discovery. The most important factor has apparently been its earlier recognition especially by the general public and those who serve in different health capacities often indirectly associated with ophthalmology. Next is the better coordination of our knowledge of its fundamentals as parts in the whole, using more efficient applications of these basic principles in the solution of often complex problems which involve the causation, progress and treatment of any glaucomatous person. In this third stage of glaucoma understanding, the more orderly and effective use of mostly previously known diagnostic and treatment methods is really responsible for our present technical progress. The better hygienic care of the glaucomatous and the preglaucomatous, the experimental use of more potent drugs and minor changes of surgical technics which summarize our newer trends of treatment are important. But even more important is that future progress will largely depend on the solution of glaucoma problems through the application of basic principles which apply to this disease as a whole, rather than as individual problems largely independent of the fundamentals on which the condition as a whole depends.

In conclusion, the more potent drugs and changes in operative technics are less important among the newer trends in the treatment of primary glaucoma than the fundamentals which have been mentioned. These involve:

A. The possibility of glaucoma as a cause of any symptom involving sight or pain in the eye, face or head, especially in those past forty.

B. In addition to any other examination, especially nonophthalmological, physicians should routinely compare every patient's intra-ocular tension with their own. If there is any difference or doubt the patient should be promptly examined by an ophthalmologist.

Glaucoma blindness is generally avoidable if the disease is recognized early in its course and treated with reasonable efficiency. Although the newer trends in treatment are important steps forward, let us face the fact that the glaucomatous blind will largely depend upon the cooperation of nonophthalmologist physicians who first attend the glaucomatous patient in a health capacity. If you and 100,000 other nonopthalmologist physicians will not forget the frequency and importance of glaucoma, or to apply daily the simple fundamentals in its prevention or retardation, glaucoma will cease to be ophthalmology's Public Enemy No. 1.

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ACUTE BACTERIAL ENDOCARDITIS OF SALMONELLA ORIGIN

REPORT OF CASE*

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The extreme rarity of bacterial endocarditis of Salmonella origin justifies the report of the following case, which was recently observed at Charity Hospital of Louisiana at New Orleans.

REPORT OF CASE

The history of this 52 year old white man, who was admitted to the hospital August 28, 1947, was secured with difficulty because he spoke and understood no English. So far as could be determined, he apparently had been perfectly well until five days earlier, when he had begun to suffer from "rheumatism" and fever after being caught in a heavy rain. The pain, which was localized in the neck, knees and ankles, was not associated with swelling or tenderness. Two days before admission he had developed pain in the back and chest, which had been continuous, and pain high in the epigastrium, which had been intermittent. He also had suffered from anorexia and malaise, and from a slight, nonproductive cough. The day of admission to the hospital he passed a stool described as "black;" this was the first gastrointestinal irregularity. A "heavy" pain in the neck appeared the same day.

Inquiry into the past history was not revealing. The family history was noncontributory. The patient was a fisherman, who lived in the dock area, where there were "lots of rats." For some time he had suffered from headaches and from occasional "gas" pains. There was a recent history of nocturia (twice nightly) and of increased diurnal frequency. He had had bilateral hernias for forty years. No history of an insect bite could be secured.

Physical examination: A man of about the stated age who seemed chronically rather than acutely ill. Temperature 102° F., pulse 126, respiration 26, blood pressure 110/52. The face was flushed, the skin was dry and hot, and the eyes were bloodshot. Funduscopic examination revealed arteriovenous nicking and an increase in the light reflex of the arteries. The nasal mucosa appeared somewhat redder than normal. The few teeth still in situ were dirty and carious.

The chest was barrel-shaped. Loud, moist, bubbling rales were heard over both bases posteriorly and crepitant rales were heard over the midlung fields. There was no change in the voice sounds. The percussion note was resonant to hyperresonant. The heart was not clinically enlarged. The sounds were distant but could be clearly heard. A grade 2 aortic systolic murmur was present. A pericardial friction rub could be heard parasternally in the fourth left interspace anteriorly. The radial pulse was full, bounding and regular. All peripheral blood vessels were thickened.

The abdomen was moderately distended and tympanitic. Peristaltic sounds were decreased. No organs or masses were palpable. Bilateral irreducible inguinal hernias the size of grapefruit were present. The prostate was moderately enlarged. Several internal hemorrhoids were present.

Except for enlargement of the right epitrochlear lymph node, no lympadenopathy was observed. The patellar reflex on the right was decreased. All other reflexes were physiologic.

Initial Laboratory Data: Urinalysis showed a trace of albumin, 1 plus sugar, and 3-4 white blood cells and 1 red blood cell per high power field (centrifuged specimen); the specific gravity was 1.012. Blood study showed 12.6 gm. per cent of hemoglobin; 14,850 white cells per cubic millimeter; 88 per cent polymorphonuclear leukocytes, 8 per cent lymphocytes, and 4 per cent immature cells. The hematocrit reading was 37 mm. and the sedimentation rate 32 mm. per hour (Wintrobe). The Kline and Kolmer reactions were negative.

Clinical Course: The patient's clinical course can conveniently be summarized in three episodes, the first dating from admission (August 28) through September 19, the second from that date through September 29, and the third from that date until death, October 11.

First Episode: The tentative diagnosis on admission was bronchopneumonia and possible typhus fever. The day after admission the patient, who had not been jaundiced when he was first examined, was definitely jaundiced. The liver, which had not been palpable at the first examination, was now palpable two finger-breadths below the costal margin and was slightly tender. There was dullness over the lower third of the right posterior lung field and more rales were present than on the

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previous day. The pericardial rub, which was still clearly heard, was explained on the basis of a pleuropericardial reaction to pneumonitis. Roentgenograms showed a suggestive enlargement of the cardiac shadow, with infiltration radiating out from the hila, more markedly on the right side and toward the right base. The roentgenographic findings were considered compatible with congestive cardiac failure. Infectious hepatitis and Weil's disease were now considered as diagnostic possibilities.

September 1, the fourth day of hospitalization, the patient had twelve liquid yellow stools during a six-hour period. Examination showed no ova, parasites or cysts, and no pus or blood. By September 4 the stools had become clay-colored and a diagnosis of carcinoma of the ampulla of Vater was briefly entertained, on the ground that the jaundice was of the obstructive type. During this episode the urine contained bile but was negative for urobilinogen in 1/10 dilution. The icterus index reached a maximum of 44.5, then fell slowly to 20.8. The van den Bergh direct reaction was 7.6, September 9, and forty-eight hours later was 12.8. The white blood cells ranged between 15,120 and 9,420 per cubic millimeter. Agglutinations were negative for typhoid (O and H), typhus fever and brucellosis, and two blood cultures were reported sterile. Electrocardiogram was normal (as was a second electrocardiogram on September 29). A study of the gastrointestinal tract with a barium meal revealed no abnormalities of any kind, including no widening of the duodenal loop. The alkaline phosphatase and the serum proteins of the blood were normal.

The patient's temperature during this first episode reached 104.6° F., fell slowly to 99.2° F. on the eighth and ninth days of hospitalization, and then rose again to 102° F. almost daily. The pneumonitis present on admission cleared up within five days, the pericardial friction rub disappeared about the same time, and by September 19, the twenty-third day of hospitalization, clinical jaundice also disappeared. At no time, in spite of the high fever and other manifestations, did the patient seem seriously ill. Two courses of penicillin were given, the first amounting to 480,000 units and the second to 660,000 units. Both courses were discontinued because there was no apparent effect from the medication. The administration of digitoxin was begun on the third day of hospitalization and was continued until death. Otherwise treatment consisted of a salt-free, high-protein, high-carbohydrate, low-fat diet, supplemented by high dosages of multivitamins and by amino acids.

Second Episode: During the second episode of his illness, which lasted for the ten-day period ending September 29, the patient was clinically free of jaundice; the icterus index was 10.4. The urine was repeatedly negative for bile and was positive for urobilinogen in dilutions ranging from 1/10 to 1/40. The white blood cell count was 18,000 per cubic millimeter on one occasion and 21,840 on another; each count was made shortly after a chill. The stools on two occasions showed larvae of *Strongyloides stercoralis*. Agglutions were negative for typhoid O and H, paratyphoid A and B, brucellosis, tularemia, and Proteus X 19. Smears were negative for malaria. Roentgenograms of the chest showed an elevation of the right leaf of the diaphragm, with the cardiothoracic ratio within normal limits. Amebic abscess of the liver was now considered a possibility. Fluoroscopy, however, revealed both diaphragms normal in position and moving freely and equally with respiration; the costophrenic sulci were clear.

During this second episode the temperature varied from 100 to 102.6°F., usually being nearer the higher level. The patient had, as noted, two chills, after the second of which blood culture was reported positive for Salmonella.

Third Episode: September 30 the patient again became jaundiced and the liver again became palpable about 3 cm. below the right costal margin; it was soft and not particularly tender. There was resistance of the right rectus muscle in the upper quadrant and the epitrochlear lymph nodes were enlarged but otherwise the physical findings were normal. There was, however, a peculiar sour odor about the patient, the so-called fetor hepaticus, which is usually associated with advanced hepatic degeneration.

The icterus index was 17.8. The urine was negative for bile and positive for urobilinogen in dilutions of 1/10 to 1/40. A stool culture was negative. The white blood cell count was 21,040, with 80 per cent polymorphonuclear leukocytes.

October 2 the blood culture was again reported positive for Salmonella, and although the organism was resistant to streptomycin in vitro in a concentration of 100 mcgm. the use of the drug was begun in dosages of 4 gm. daily and was continued until 20 gm. had been given. There was no perceptible effect on the temperature elevations, which ranged from 101 to 103° F., with one elevation to 105° F.

October 9 the patient had a severe chill and his temperature rose to 104.4° F. For the preceding forty-eight hours he had complained of considerable "gas", and hiccoughing suggestive of diaphragmatic irritation. October 11 he became nauseated early in the morning and vomited several times. His condition seemed good, however, and he did not complain of pain. In the evening he went into a state of shock. He vomited dark fluid, which grossly did not contain blood, and he complained of occasional pain across the lower portion of the abdomen, which was tympanitic and rigid. The liver was no longer palpable but liver dullness could be demonstrated immediately below the right costal margin. The mucous membranes were pale and the skin was a curious slate-color. Petechiae were present over the flexor surfaces of the forearms.

Death occurred suddenly, while an infusion was being given.

The clinical diagnosis was infectious hepatitis and Salmonella septicemia.

Necropsy: Necropsy, which was performed ninety minutes after death, revealed the following data pertinent to this discussion:

The body measured 167 cm. and weighed 122 pounds. Petechiae were present over the flexor surfaces of both forearms.

The peritoneal and pleural cavities were essentially negative. The pericardial cavity was obliterated by fine fibrinous adhesions which were extremely friable and separated easily. The heart weighed 405 mg. The valve measurements were as follows: TV 12.5 cm.; PV 7.5 cm.; MV 11 cm; AV 8 cm. The myocardium of the right ventricle measured 0.4 cm. and that of the left measured 2.5 cm.

The epicardium was covered with very fine fibrinous adhesions. The myocardium was essentially normal. The coronary system presented moderate arteriosclerosis, the lumen in some areas being reduced to approximately 60 per cent of its normal diameter. The first portion of the aorta presented early calcific changes, with some encroachment on the cusps of the aortic valve. On the pulmonary cusps were verrucous, friable vegetations, each measuring 0.7 by 0.5 by 0.5 cm. They were tannish-red mottled with pink and crumbled upon gentle pressure. A slightly concave area on the terminal portion of the left semilunear pulmonary valve vegetation indicated that a part of this vegetation was missing. Fenestration of the cusps was present; the largest defect measured 0.3 by 0.2 cm.

The aorta showed marked atheromatous changes, with calcification and ulceration in the iliac region.

The right lung weighed 510 gm. and the left 400 gm. For the most part the lungs were semicrepitant and congestion was moderate. In the left lower lobe was an area of infarction 6 by 5 by 5 cm.; the artery leading to this area was completely occluded. In the right pulmonary artery was a large, recently formed thrombus which practically occluded the vessel. No infarcts were observed in the right lung.

The spleen weighed 210 gm. Its appearance was that of acute splenic tumor. Congestion was marked. The consistency was firm and the capsule did not wrinkle when the organ was placed on a flat surface. A slight perisplenitis was present, as evidenced by a thin layer of fibrinous material. The liver, which weighed 2,100 gm., presented no gross abnormalities other than chronic passive congestion. In the distal 2 feet of the colon were numerous hemorrhagic ulcers, each measuring approximately 1.5 by 1 cm. These ulcers, which were covered with bloody mucus, did not appear to penetrate the mucosa.

The brain, bladder, kidneys, genital organs, gallbladder, pancreas, adrenal glands, organs of the neck, spinal cord, bone marrow, and middle ears were grossly normal.

Microscopic Examination: Examination of the heart showed a fibrinous exudate with round cell infiltration over the surface of the pericardium. The myocardium was essentially normal. Attached to the cusp of the pulmonary valves were large vegetations composed of acute inflammatory cells, layers of fibrin, a small amount of connective tissue, and colonies of bacteria (Fig. 1). This cusp was not covered with endothelium (Fig. 2). It was itself involved in the acute inflammatory reaction and was infiltrated with polymorphonuclear leukocytes and macrophages. The valve did not seem to have been involved in a previous inflammatory process. The coronary artery showed atheromatous changes and the lumen was encroached upon and was approximately 80 per cent of the normal diameter.

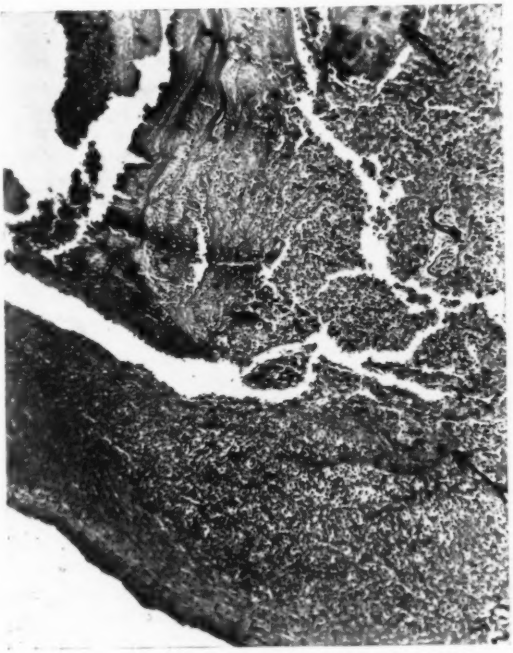

Figure 1. Section of pulmonary valve (low power magnification); arrows point to colonies of bacteria.

Sections of the lung revealed an area of infarction with edema, congestion, acute inflammatory exudate, colonies of bacteria, and thickening of the alveolar walls; numerous pigment-laden macrophages were present. A section of a thrombosed artery showed layers of organizing fibrin and blcod cells within the lumen (Fig. 3).

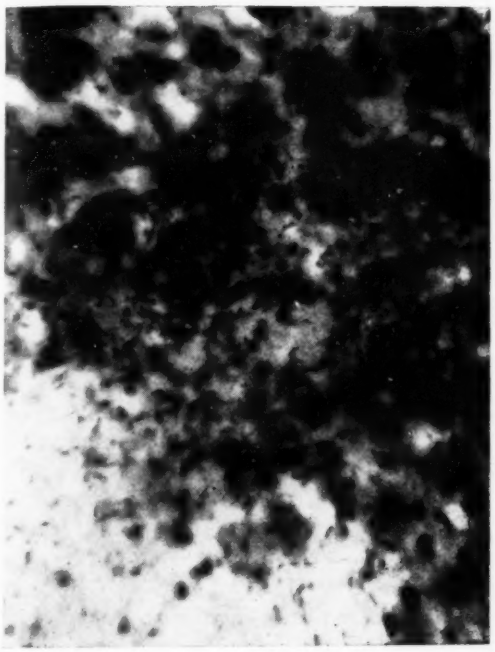

Figure 2. Section of heart valve (oil magnification) showing gram-negative rods in bacterial colonies.

Figure 3. Section of lung (low power magnification) showing septic thrombus in a pulmonary artery.

Sections of the spleen showed marked congestion with some decrease in malpighian corpuscles. The capsule was thickened and there was a fibrinous exudate on the surface.

The kidneys presented a picture of nephrosclerosis with a superimposed inflammatory process which appeared in isolated foci throughout the cortex. The process was characterized by marked congestion of the capillaries and small arterioles and by slight degeneration of the arterial walls. The glomerular pattern appeared undisturbed by this process but showed many of the hyalinized areas characteristically found in nephrosclerosis. The tubules in these focal areas showed cloudy swelling of the cells with early degeneration. (Figure 4.)

Figure 4. Section of kidney (low power magnification) showing focal nephritis.

The liver revealed chronic passive congestion. The pattern as a whole was undisturbed. A cavernous hemangioma was present.

Final Diagnosis: The essential parts of the final pathologic diagnosis were: acute suppurative bacterial endocarditis of Salmonella origin with vegetations of the pulmonary valve, fenestrations and cardiac hypertrophy; pericarditis; pulmonary thrombosis with septic infarction and congestion; toxic focal nephritis; hemangioma of the liver; chronic passive congestion of the liver; perisplenitis; ulceration of the colon.

COMMENT

The organism recovered by blood culture on two occasions was Salmonella, on the basis of morphologic characteristics and biologic reactions. The species of Salmonella at the present time number well over 150. *S. paratyphi A, B* and *C*, and *S. sendai* are

of chief importance in human infection. Specific types are identified by means of serologic tests which utilize their antigenic structure. This is impractical in the average laboratory. Typing is principally used in research as a quick and accurate method of identification of unknown cultures. Further identification of the organism in this case was not done.

This case differs in several respects from the usual case of acute bacterial endocarditis: (1) Acute bacterial endocarditis usually develops during the second and third decades and is most frequently associated with a history of valvulitis originating in childhood. In this case no antecedent history of cardiac disease could be obtained, and the disease first became manifest during the sixth decade. (2) Seventy-five per cent of all cases of acute bacterial endocarditis are superimposed upon previously deformed valves which are the result of rheumatic valvulitis. In this case there was no evidence of previous valvular lesions and no evidence of congenital valvular defects, which are another predisposing factor. (3) The valves involved in acute bacterial endocarditis are, in order of involvement, the mitral, the aortic ,the tricuspid and the pulmonary. In this case the pulmonary valve, which is least often affected, was the site of the lesion.

The presence of petechiae, the thrombotic process in the lungs, the freedom from involvement of the myocardium, the toxic process in the kidneys, and the microscopic picture generally correspond with the usual observations in acute bacterial endocarditis.

The explanation of the clinical course in this case is difficult. When the patient was first seen, the emphasis was upon the pneumonitis and upon the cardiac condition, which was assumed to be congestive failure. On the first examination the liver was not palpable and the patient was not jaundiced. Twenty-four hours later the liver was enlarged and jaundice of the obstructive type was evident. Hepatomegaly and jaundice then regressed, only to become marked again at a later date, when, however, the jaundice was of the nonobstructive variety. Postmortem examination revealed no changes of consequence in the liver, yet ten days before death a physician who saw the patient for the first time minimized the cardiac aspect of the case and thought all the emphasis should be upon the hepatic factor. The cardiac involvement, as a matter of fact, was never clearcut after the first few days. The electrocardiograms were essentially negative and the patient at no time had symptoms clearly referable to the heart.

The state of shock which immediately preceded death is probably to be explained by the pulmonary infarction found at necropsy. The origin of the infarction and of the occlusion of the pulmonary vessels can reasonably be assumed to be the vegetations on the pulmonary valve.

To date, four proved cases of acute bacterial endocarditis have been reported in which Salmonella was the etiologic agent. The case of *Salmonella suipestifer* endocarditis reported in 1921 by Andrewes and Neave[1] in a four year old child ill for two weeks with *S. suipestifer* bacteriemia is probably authentic, but since recovery ensued it cannot be included in the list of proved cases.

The first two cases of the disease were recorded by Forster[2] in 1939. In both, the clinical picture was that of a severe infectious process with endocarditis; *S. suipestifer* was isolated from the blood streams in both. The second patient received sulfanilamide in adequate therapeutic dosage, without symptomatic or other improvement. Both patients had marked disturbances of cardiac rhythm and one had auricular fibrillation for the entire three-week period of hospitalization. At necropsy distinct evidence of antecedent cardiac injury was found in both cases.

The third recorded case of *S. suipestifer* endocarditis was reported by Read,[3] also in 1939. The case differs from Forster's cases in that the organism was of the European, not the American strain. As in Forster's cases, the clinical picture was that of a severe infection complicated by endocar-

ditis; there was no apparent antecedent disease. Sulfanilamide was not useful.

The fourth case was reported in 1942 by Goulder and his associates.[4] The patient, a 58 year old woman, had a previous history of rheumatic heart disease. Blood cultures were positive for the European type of *S. suipestifer*. Sulfapyridine was of doubtful value.

In their review of the four recorded cases of Salmonella endocarditis, including their own, Goulder and his associates noted the following points: (1) In three cases the lesion was superimposed on a damaged valve; rheumatic infection was antecedent in two cases and syphilis in one. (2) Embolic phenomena were noted in three cases. (3) The spleen was never palpable. (4) Fever was high and was associated with chills in all cases. (5) Leukocytosis, an unusual finding in uncomplicated *S. suipestifer* bacteriemia, occurred in all cases. (6) Changing murmurs were noted in three cases. (7) Chemotherapy was not effective in the three cases in which it was used.

The conclusion of Goulder and his associates, that endocarditis of *S. suipestifer* origin should be suspected in any case of bacteriemia caused by this organism whenever changing murmurs, embolic phenomena and leukocytosis are associated, would not have been particularly useful in the case reported herewith, since petechiae appeared only just before death, leukocytosis was not unusually high, and no significant murmurs or other marked cardiac abnormalities were present at any time. It should be noted that in this case neither penicillin therapy nor streptomycin therapy was useful, just as chemotherapy was not useful in the three reported cases in which it was used.

SUMMARY

The case of Salmonella bacteriemia associated with endocarditis reported in this communication is apparently the fifth case of acute bacterial endocarditis of this origin to be recorded. It differs from other recorded cases in that the cardiac aspects of the illness were throughout of an extremely insignificant character. Neither penicillin nor streptomycin altered the clinical course.

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POLYNEURITIS WITH FACIAL DIPLEGIA SYNDROME DEVELOPING DURING ANTIRABIES VACCINATION

VINCENTE D'INGIANNI, M. D.
AND
I. L. FONTENELLE, M. D.
NEW ORLEANS

Polyneuritis with facial diplegia is a well known clinical entity. It has been described in the literature under a variety of terms. This condition has been referred to as Guillain-Barré syndrome, acute polyneuritis, infectious polyneuritis, motoneuritis, acute polyneuritis with facial diplegia, and acute ascending paralysis. In addition, several other descriptive terms are used for the same condition.

The cases which come under the above terms are characterized clinically by an acute onset, mild or no febrile reaction, radicular neuritis, cranial nerve palsies, muscle tenderness and certain cerebrospinal fluid changes.[1] One of the commonest features of facial diplegia with polyneuritis is the albuminocytological dissociation. The occurrence of a normal cell count and an increase of the fluid proteins is a constant finding.[2] The course of the disease is usually benign and recovery requires from six months to two years. Death may occur from respiratory paralysis, intervening sepsis, or general debilitation.[1]

The etiology of Guillain-Barré syndrome, as we shall choose to call it in this report, is unknown. The syndrome is preceded by mild infections in the majority of cases. Kamman and Weisberg[2] and Morris[3] reported cases following serum sickness resulting from tetanus antitoxin. Chusid and

Marquardt[4] reported a case following exposure to mustard gas. Garvey, Jones and Warren[5] reported 6 cases which followed the use of sulfanilamide and general hyperthermia.

The syndrome must be distinguished from other infectious diseases of the central nervous system as well as other types of polyneuritis. The chief entities to be differentiated are acute anterior poliomyelitis and diphtheritic polyneuritis. Progressive involvement, sensory changes, symmetrical ascending paralysis, and albuminocytologic dissociation in the cerebrospinal fluid favor the diagnosis of Guillain-Barré syndrome. Diphtheritic polyneuritis may be ruled out by the presence of a positive Schick test, the failure to find the pathogenic bacteria in nasopharyngeal smears and culture, and the absence of clinical signs and symptoms of diphtheria.[1]

Paralytic reactions resulting from antirabic vaccine occur infrequently. In a statistical analysis and review of the reports from Pasteur Institutes throughout the world up to 1927, Remlinger[6] reports 329 cases of paralytic accidents occurring in 1,164,264 inoculations. This is an incidence of 0.028 per cent. In 1938 McKendrick[7] reported 139 cases of paralysis in 755,891 treatments from 1927 to 1938. This is an incidence of 0.017 per cent.

In general three types of paralytic reactions are reported following antirabies vaccination: (1) Ascending paralysis of Landry, (2) dorsolumbar myelitis, and (3) neuritis or isolated peripheral nerve paralysis. The ascending paralysis is 30-40 per cent fatal, and the myelitic type is about 5 per cent fatal. No fatalities are reported in the peripheral nerve type.

In addition to the above, Horack[8] reports several types of skin reaction following antirabies vaccination: (1) Generalized urticarial reaction; (2) delayed reactions of the tuberculin type occurring at the site of the injection and characterized by local redness, induration, tenderness, and itching; (3) reactions similar to the tuberculin type, but more severe and frequently associated with constitutional reactions. This latter group should be closely observed for signs of development of paralysis.

The case herein presented deals with an individual who was receiving a second course of antirabic serum, and who developed a typical ascending neuritis. His neurological condition, at its onset, was accompanied by itching and a rash, along with generalized weakness of all extremities. This latter condition finally forced him to discontinue the antirabic treatment. The disease took on the typical attributes of an ascending paralysis with the spinal fluid picture seen in polyradicular neuritis, the relatively normal cell count and the markedly increased protein level. The patient made a complete recovery in twelve weeks.

CASE REPORT

Mr. Y., aged 38, was bitten by a rabid dog on October 17, 1947. On October 20, he began antirabic therapy daily until October 30. On October 27, he noticed itching and the sensation of "pins and needles" in all extremities. On October 28, his legs became weak and wobbly; he noted an inability to put his hands on objects reached for. His leg weakness became progressively worse, and he was not able to make more than one step without falling. At this time he began to have severe pains in the calves of his legs. It might be well to mention here that the patient had received a course of antirabies treatment one year previously that was uneventful.

Mr. Y. was admitted to the hospital on October 31, 1947. There were no noteworthy findings except in the neurological examination. The report was as follows: Positive past pointing. Bilateral absence of reflexes in the lower extremities. Positive bilateral Kernig sign. The only significant laboratory findings were in the spinal fluid.

Spinal fluid findings on admission: Sugar, 67.0 mgm.; Wassermann, negative; globulin, 2 plus; protein, 230.0 mgm.; chloride, 660 mgm.; colloidal gold curve, 0000224210; polys, 5.

On November 6, 1947, the spinal fluid was reported as: Sugar, 94.0 mgm.; Wassermann, negative; globulin, 3 plus; protein, 280.0 mgm.; colloidal gold curve, 000024543211; polys, 9.

Blood count: Red blood cells, 5.54 million; white blood cells, 10,350; hemoglobin, 128%; polys, 77; lymphocytes, 23.

PROGRESS NOTES

11/2/47 Patient developed left facial paralysis.

11/3/47 There was a weakness on the right side of the face. The back of the neck and the lumbar area were extremely painful and tender.

11/4/47 There was complete facial diplegia, and inability to move the lower extremities.

11/6/47 The patient exhibited an inability to move the left upper extremity. The deep reflexes were absent. There was no urinary or rectal incontinence.

11/11/47 His complaints included dyspnea, loss of appetite and pains in the thighs, which were relieved only by narcotics.

11/22/47 The patient became constipated. Fecal impaction removed. The maximum temperature was 100° F., pulse 100, respirations 28 per minute.

11/24/47 The patient was discharged with the diagnosis: Polyradiculoneuritis.

CONCLUSION

An early evaluation of the findings in this case might well have resulted in holding the antirabic serum responsible for the accompanying paralysis and other symptoms. However, the spinal fluid findings definitely fixed the diagnosis as Guillain-Barré syndrome.

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NEW ORLEANS
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LIVER FUNCTION AND CHOLINE

The theory has been evolved in recent years that liver dysfunction, fatty infiltration and cirrhosis are part of a sequence resulting from failure of adequate nutrition. The concept of what is proper nutrition is a broad one which is conceived to include suitable proteins, carbohydrates and fats together with sufficient vitamins and "lipotrophic" substances. The term lipotrophic refers to those agents, principally choline, which appear to assist the liver in removing fat.

The etiology of cirrhosis is unknown but a number of contributing factors are thought to bear on the problem. Nutritional deficiencies conceivably lower the local resistance to infectious disease and to toxic chemicals (such as mercury, arsenic, carbotetrachloride, chloroform, ether, gold and sulfonamides.) In whatever manner the process is initiated, atrophic cirrhosis is characterized by diffuse fatty infiltration with the overgrowth of fibrous tissue appearing later—presumably as a result of cell destruction. Alcohol in some individuals contributes to the picture by promoting a dietary deficiency and possibly is a factor in producing fatty liver. As has been well understood in the past, the sclerosing effect of fibrous tissue then prevents proper circulation within and through the liver, ultimately bringing the terminal picture of cirrhosis as it is currently recognized.

While disease of the liver is being slowly produced in this fashion. dysfunction is also partially manifest. It is clear that the liver has many functions and these concern the elaboration of products of fats, carbohydrates and proteins ingested. The liver is also concerned with formation and clotting of blood; the regulation of blood volume; the immunity and reticulo-endothelial defense system; the detoxification of certain harmful substances and many other processes of which we have even less knowledge.

There is an intimate connection between liver and kidney function. In Weil's disease there is a rapid appearance of azotemia. In experimental diets without choline there develops a picture comparable to uremia. Both organs are adversely affected in acute infectious diseases and in toxemias of pregnancy.

The manner in which choline works in the liver physiology is not clear. In the biochemistry of the recent past, choline was a toxic substance derived from protein. It is felt now that it is concerned in cellular metabolism stimulating increased utilization of fats by the hepatic cells. It is also thought to mobilize neutral fat by promoting the formation of choline-containing phospholipids. The use of choline in conjunction with the proper dietary regime has been shown in human livers to change

a fatty liver to one containing the normal amount within four weeks. It has also been reported to improve certain megaloblastic anemias resistant to liver therapy. The bearing that choline has on cholesterol metabolism and arteriosclerosis is obscure. It is reported that it has an effect in returning high plasma lipids to normal levels, but Best states the evidence that choline prevents cholesterol atherosclerosis is at present worthless.

The early signs and symptoms of liver dysfunction are not definite. They consist of vague dyspepsia, ill defined abdominal discomfort leading to pain, nausea, particularly in the morning, flatulence and eructations. Following later are persistent asthenia, anorexia, loss of weight and anemia. When hepatic cirrhosis is moderately advanced, one finds vascular spiders, edema of the ankles, lowered serum proteins, ascites and hemorrhoids. Jaundice may be noted at any stage but is expected in some degree in the terminal phases.

The usefulness of the new concepts of liver disease is to delay and possibly arrest the development of the above picture. The therapy suggested in the light of these observations is dietetic and medicinal. In the stage of ascites and edema salt is restricted or eliminated. Fat in the diet is reduced to about one gram per kilo; proteins are increased to two grams per kilo and carbohydrate sufficient for caloric requirement is advised. Vitamin concentrates are necessary adjuncts to the diet. The lipotrophic agents are given partly through the proteins of milk and cheese but primarily supplied by choline. The amount advised is two to three grams of a choline salt per day. Weekly injections of liver extract are desirable.

The results in favorable, that is early cases, are gratifying. Ascites and edema have been seen to disappear; appetite usually increases. There may be a gain in weight. There is usually an increased sense of comfort amounting to well being. In advanced cases of cirrhosis the results are less satisfactory but in many instances the change is sufficient to give comfort.

The knowledge that possibly improvement may follow treatment in early cirrhosis will increase the vigor of attack on the problem. Many individuals may be spared the terminal picture.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

STATE SOCIETY AWARDS

At the opening session of the 1949 Annual Meeting special recognition was given members of the State Society who have practiced medicine for fifty or more years and also to the General Practitioner of the Year for the State of Louisiana, selected in 1948. Following are talks presented by the President of the State Society, Dr. M. D. Hargrove, at the time of presentation of lapel pins to the Fifty-Year Doctors and a plaque to Dr. Charles M. Horton, recipient of the General Practitioner Award.

FIFTY-YEAR DOCTORS

During the past year it was decided that the Louisiana State Medical Society should give special recognition to those doctors who have been in practice for fifty years, and are still members of the State Society, Accordingly, Dr. Talbot, our Secretary-Treasurer reviewed the roster of our members, finding among them 46, who have been practicing for fifty years and are still and general practitioners. Some names are members of the State Society.

The list contains the names of specialists

familiar in medical circles throughout the world and other names that are simply known in the community where the doctor has labored through the fifty years of practice. Regardless of the extent of their fame, the names are of men who through fifty years have given to their patients and to medicine the best there was in them.

Medicine and the doctor occupy a unique and honored place in most American households. That position of esteem and respect is due to the devotion to duty and sterling character of the family physician as portrayed by these 46 doctors. They, more than any others, have really put a heart in the practice of medicine. Truly they are deserving of this tribute to the American doctor.

Who shall measure devotion, or put a price on sacrifice?
Who shall assess the long war against the power of Death?
Or set a sum upon the gift of Life?

There is a service beyond the measure of a fee.
A cause above remuneration.
An ideal for which there is no price.
This is the service . . . the cause . . . the ideal . . . of the American doctor.

Who shall reckon it, and by what formulae?
How much for the laughter of a little child rescued out of crisis?
What's the cost of discouragement?
Who can pay for a sleepless night?
Name the price of a cure?

There is no algebra for it, no scribble of figures, no proper value.
For this is a service as large as life, and as manifold.
It is a soldier crying in agony on a thousand battlefields.
It is the terrible word "Why?" under the surgeon's probe.
It is the end of pain.
It is Hope.
It is the lonely unending quest for knowledge.
It is the fight against ignorance, sloth, superstition.
It is the dumb unspeakable joy in the eyes of a parent.
It is the rock of grief.
It is cold rain and pounding storm and bone-weariness, and the new born babe gasping its first breath in the grey dawn.
It is all this and the quiet glory of the job done,
Dedicated to service—in the name of Mercy And the common brotherhood of man.

We are proud of you my friends, and of your record of service. The Louisiana State Medical Society is distinctly honored by your fifty years of service. It has been a job well done and we can only hope that you will be with us and those you serve for many more years.

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GENERAL PRACTITIONER

The Louisiana State Medical Society is tonight honoring and being honored by a distinguished member of our society.

You are aware of the policy of the AMA in selecting a general practitioner, who, because of the quality of his work, preferably in a rural community, is to be designated as the General Practitioner of the year. In 1948 each state society was requested to submit the name of one member whom they thought deserving of that honor. From those submitted the Board of Trustees of the AMA selected three, presenting their names and their qualifications to the House of Delegates, who by ballot, selected one of the three doctors for the award.

The award was first made in 1948 to Dr. Archie Sudan of Colorado. The Louisiana State Medical Society did not present a candidate that year. Last year we asked the various parish societies to suggest a member and give his qualifications. From those suggested the Executive Committee selected Dr. Charles M. Horton as the General Practitioner of the Year for the State of Louisiana.

His name was presented to the AMA and he was selected by the Board of Trustees as one of the three to be presented to the House of Delegates. Of the three, Dr.

Priestley of South Carolina, was selected as the General Practitioner of the Year, but being one of the three presented to the House of Delegates was a distinct honor to Dr. Horton and to Louisiana.

Many of you know Dr. Horton and the splendid job he has done in the practice of medicine. His life and work reads almost like a story book. For 37 years he has served the people of Franklin as their family physician. The quality and character of that work has won for him a place in their hearts which cannot be added to or detracted from. They know how well he deserves any and all honor that he has received.

Through the years of his practice Dr. Horton has been, and continues to be, one of the most active members of the Louisiana State Medical Society. He has held practically every office, served on various committees and worked unceasingly for the advancement of medicine.

In recognition of the selection of Dr. Horton as the General Practitioner of the Year for the State of Louisiana, and his selection by the Board of Trustees of the AMA as one of the three outstanding practitioners of the nation, the Louisiana State Medical Society presents Dr. Horton with this plaque.

Dr. Horton, presenting you with this plaque is an unusual privilege and honor coming to me as President of the Louisiana State Medical Society. We are being honored by you. The work you have done over the past 37 years speaks for itself and should serve as an example to the doctors of today.

On behalf of the State Medical Society I present you with this plaque, as an expression of our esteem and in recognition of the honor bestowed upon you, and by you upon the Louisiana State Medical Society.

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LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

CANCER CONTROL ACTIVITIES

Tulane and LSU have in operation Cancer Detection Clinics. No additional clinics of this type will be planned but these two will be continued for teaching purposes.

The two universities have agreed to instruct eight physicians a week. Invitations to attend these Detection Clinic courses will be sent out from our State Medical Society office. Be on the alert for your invitation—we feel it will be well worth your while.

The Cancer Committee of the State Medical Society is planning a year-round program of postgraduate education. Twice during the year—Fall and Spring—a symposium on the diagnosis and treatment of cancer will be presented in each of the eight congressional districts.

Tulane and LSU will furnish the personnel for these courses. The expense will be defrayed by monies received from the Louisiana Division of the American Cancer Society, the State Department of Health, and the Louisiana State Medical Society.

Make plans now to attend these courses. Let's not be content to keep pace with the lay educational programs—let's stay ahead.

JOHN G. SNELLING, M. D., Chairman
Executive Committee
State Medical Society Cancer Committee

EAST AND WEST FELICIANA PARISH MEDICAL SOCIETY

The Bi-Parish Medical Society met with Dr. L. F. Magruder and staff in the East Louisiana State Hospital. This was a dinner-meeting after which a scientific program was presented.

Dr. Paul Jackson presented an excellent paper on Spontaneous Pneumo-Hemothorax. Dr. C. S. Toler led the discussion.

At this meeting Dr. L. F. Magruder was elected a member of the society. A vote of thanks was extended to Dr. Magruder and the staff and employees of the East Louisiana State Hospital for their excellent arrangements.

The society adjourned to meet again in the East Louisiana State Hospital on the first Wednesday in Septembzer, 1949, at 7:30 P. M.

AMERICAN HOSPITAL ASSOCIATION

A six-day course especially designed for hospitals in the planning stage has been added to the 1949 American Hospital Association Institute schedule.

The Institute on Hospital Establishment will be held on the Chicago campus of Northwestern University from August 8 through August 13.

American Hospital Association officials say the course was set up to meet a need that seems particularly acute in localities which have not heretofore operated a hospital.

The curriculum is being developed in cooperation with the Hospital Services Branch of the United States Public Health Service's Hospital Facilities Division.

Among topics to be taken up are establishing a budget, determining patient charges, personnel problems, public relations responsibilities, setting up a medical staff and nursing organization, service departments and business procedures.

Applications for attendance at the Institute should be addressed to Mr. Roy Hudenburg, American Hospital Association, 18 East Division Street, Chicago 10, Illinois. Tuition fee is $35. Reservations can be made for dormitory accommodations on the University's campus.

Enrollment in the Institute on Hospital Establishment is limited to administrators of hospitals in the planning stage, members of governing boards of such institutions, and representatives of hospitals that are members of the American Hospital Association.

POST GRADUATE MEDICAL ASSEMBLY OF SOUTH TEXAS

The Fifteenth Annual Meeting of the Post Graduate Medical Assembly of South Texas will be held at the new Shamrock Hotel in Houston, November 29-December 1, 1949. Sixteen out-of-state distinguished medical guest lecturers will appear on the program, representing the various specialties of medicine. Physicians planning to attend this meeting are urged to contact the Shamrock Hotel or the hotel of their choice as soon as possible for hotel accommodations. Further information may be obtained by writing the Executive Secretary at 229 Medical Arts Building, Houston, Texas.

NEWS ITEMS

Dr. Roy Carl Young of Covington attended the American Psychiatric Meeting in Montreal, Canada, May 22-27, 1949.

While there he also attended the Meeting of the National Association of Private Psychiatric Hospitals and was elected Treasurer of this organization for a period of one year, as well as to the Board of Trustees for a period of three years.

Dr. T. A. Watters was a guest speaker at the annual session of the Mississippi State Medical Association in Biloxi on May 10. His subject was "Common Psychiatric Problems and Psychosomatic Disorders in General Practice."

RESEARCH FELLOWSHIPS—THE AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians announces that a limited number of Fellowships in Medicine will be available from July 1, 1950-June 30, 1951. These Fellowships are designed to provide an opportunity for research training either in the basic medical sciences or in the application of these sciences to clinical investigation. They are for the benefit of physicians who are in the early stages of their preparation for a teaching and investigative career in Internal Medicine. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the proper pursuit of his work.

The stipend will be from $2,200 to $3,200.

Application forms will be supplied on request to The American College of Physicians, 4200 Pine Street, Philadelphia 4, Pa., and must be submitted in duplicate not later than October 1, 1940. Announcement of awards will be made November, 1949.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

WOMAN'S AUXILIARY
THE CONVENTION OF THE AMA AUXILIARY

Dear Auxiliary Members:

Instead of sending the usual message through the Journal at this time, I thought it might be interesting to tell of some of the happenings at Atlantic City.

As you strolled along the boardwalk and saw men of the medical profession and their wives greeting each other, you had a glowing feeling inside to see people so closely allied, all with the same objectives and fighting for the same ideals. There are few dissenters among the ranks!

The Auxiliary made a fine showing this year; 1500 women registered, the largest yet to attend a National Medical Convention. Did you know that the total membership is 49,000?

Forty-eight states were represented; nineteen state presidents came and the reports of forty-two states were read.

At the luncheon in honor of Mrs. Kice, National President, I sat next to a Miss Lam of Honolulu, whose mother is president of the Hawaiian Auxiliary. A doctor from the islands sent orchids to all the ladies present as a friendly gesture. Flowers were everywhere. At this luncheon, Mrs. Kice introduced her daughter and son-in-law, both doctors from Long Island.

At the luncheon in honor of the past presidents, we had the good fortune to hear both Whittaker and Baxter. The latter is Leone Baxter Whittaker, the wife of Clem Whittaker, who sent such informative literature from the Educational Campaign. Leone Baxter spoke for one hour on the evils of compulsory health insurance and the way to combat it. She held our attention throughout with her brilliance and personality.

Both Dr. Sensenich, President of the AMA, and Dr. Irons, President-elect, addressed us at the Wednesday luncheon. We appreciated the fact that such busy men would take time out to speak to us.

Many interesting things were brought out at the meetings. Oklahoma presented a document attested to by a Notary Public that in 1907 it had formed the first medical auxiliary. This year sees the first woman doctor to be president of a medical association. She is a charming woman! Maine is the baby auxiliary, not quite a year old. Oregon has an auxiliary member, the wife of every doctor who is a member of the Medical Association. Louisiana had two "firsts" among the States, the first permanent Rural Health Chairman and the first Auxiliary to the AAGP.

There were round-table discussions at which it was interesting and enlightening to get the viewpoint of the various auxiliaries. These discussions were on legislation, program and public relations. Because of the illustrated circulars sent out by Mrs. Yaguda, Public Relations Chairman, her panel was the best attended. Note bene!

Now comes the best news of all! I am proud to report that Louisiana's own Mrs. Arthur Herrold of Shreveport was elected without opposition and with much enthusiasm to the office of National President-elect. None deserves it more than she, and can fill it more efficiently. Our best wishes to her.

I think it best that I stop now as anything else I may say would be an anti-climax.

Agnes Berdami Dunn, President
Woman's Auxiliary to the Louisiana
State Medical Society

P. S. Be sure to read the July "News and Views" for the suggested program of the State Auxiliary for 1949-1950.

A. A. G. P. AUXILIARY ACTIVITIES

Surpassing all expectations in achievement and enthusiasm, the Woman's Auxiliary to the Louisiana Academy of General Practice held business and social sessions coincidentally with the men's groups on May 7 and 8 in New Orleans.

Highlighting the business session, Mrs. Joseph W. Kelso of Oklahoma City, Okla., president of the Woman's Auxiliary of the Southern Medical Association, was most helpful with suggestions concerning the various articles of the proposed constitution—advice garnered from years of experience in organizing women's groups. Most encouraging too were Mrs. Kelso's views on the advisability of the Louisiana group advancing plans for the organization of a Woman's Auxiliary to the American Academy of General Practice. Seconding the counsel of Dr. J. P. Sanders, first president of the Louisiana group (and one of the original thirteen who conceived and planned the American Academy of General Practice, Mrs. Kelso urged the need for such a unit and its unestimable possiblities for far-reaching influence.

Special guest of honor at the social functions was Mrs. Elmer Clinton Texter, wife of the president of the American Academy of General Practice. Other Auxiliary members of national note feted at the Patio Party honoring Mrs. Emma Wigginton, president of the first auxiliary to a state chapter of the American Academy of General Practice, were Mrs. Luther H. Kice, and Mrs. Arthur Herold, president and treasurer of the Woman's Auxiliary to the American Medical Association. Perfect in every detail of appointments and service, the Patio Party represented the painstaking planning of Mrs. George Feldner of New Orleans, and her convention committee of women from the first and second districts of the state. Dr. and Mrs. Feldner also entertained at a pre-convention cocktail party honoring their friends the Wiggintons, and a number

of early arrivals for the convention.

At the disposal of the A. A. G. P. Auxiliary members, a suite located in the Roosevelt Hotel proved a thoughtful arrangement for the convenience of both in and out-of-town members attending the sessions of the convention which extended from early morning until evening when a combined dinner and lecture meeting with the husbands climaxed the activities.

With the gracious charm with which she presided throughout the convention, Mrs. Wigginton installed the new officers of the Woman's Auxiliary to the Louisiana Academy of General Practice. They are: Mrs. Dorman B. Barber of Alexandria, president; Mrs. George D. Feldner of New Orleans, president-elect; Mrs. John W. Atkinson of Gretna, vice-president; Mrs. N. M. Bryan, Jr., of Alexandria, secretary; Mrs. Frank H. Davis of Lafayette, recording secretary, and Mrs. Esmond A. Fatter of New Orleans, treasurer.

Appointment of councilors and permanent chairman will be announced shortly and members and prospective members will be contacted for local meetings preparatory to activities of great interest preceding the national convention scheduled at St. Louis in the Spring.

Mazie Adkins Guidry
Chairman of Publicity

BOOK REVIEWS

A Textbook of Clinical Neurology with an Introduction to the History of Neurology: By Israel S. Wechsler, M. D. 6th ed., Philadelphia, W. B. Saunders Co., 1947, pp. 829, illus. Price, $8.50.

The latest edition of this neurological standby is as usual a reference to have within arm's reach of those of us whose work makes it pertinent. It should certainly be a vade mecum for the medical student. Nevertheless, this reviewer considers it too sketchy in parts in spite of the desirable limitations on text-book size. Alzheimer's disease and its diagnostic differential Pick's disease are most certainly not rare entities of diagnostic consideration and yet the former is covered in 43 lines and the latter in 17! Electroencephalography is mentioned only in passing, particularly in the discussion of brain tumors and of the convulsive disorders where the technique is preeminently useful. No mention is made of the psychodynamic aspects of early multiple sclerosis although the either-or diagnosis of neurosis vs multiple sclerosis is common in some circles. Postencephalitic chorea is not discussed but simply cited to exist, whereas the almost preciously rare Dubini's chorea and the dubious chorea of Bergeron and Henoch are given each a paragraph. Some 57 pages which could have been devoted to more extended comment on neurology, or inclusion of omitted data of the type cited above, are devoted to an archaic Freudian presentation of the neuroses made notable by such statements as "Of all methods, psychoanalysis alone offers a consistent psychological approach . . .", and that dessicated old analytic chestnut "—many of Freud's opponents and lip-worshipping adherents adopt nearly all of his concepts, then berate him for refusing to abandon *that which their own resistance forbids them to accept.*" (Underlining mine—HTP). This section also contains a curious pre-Kinsey comment on the perversions. It would seem worthy of question whether a large section on the neuroses has any place in a textbook of neurology. The author well states in his introduction, "No neurological status is complete without a mental examination," but references could well have been made to standard texts for the generally accepted classifications of mental disorder, a useful mental status examination outlined, and abnormal psychology theory avoided altogether. Certainly the medical student, the practitioner and the psychiatrist would benefit were more space taken up with the "why" of the sign, reflex, etc., instead of just the "where" and the "how." Despite Wartenberg, it is noted that Wechsler continues to speak of periosteal reflexes.

H. Tharp Posey, M. D.

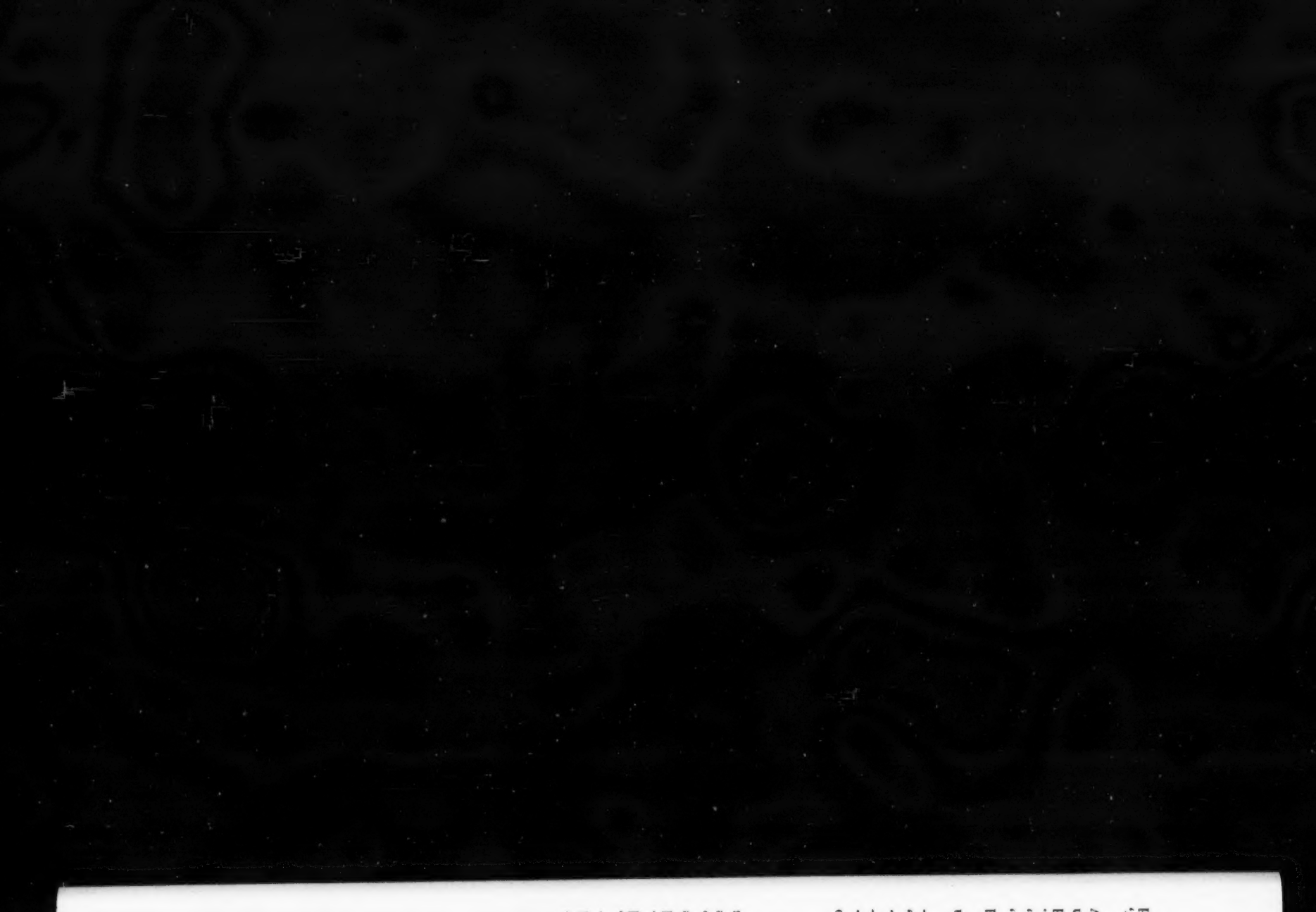
PUBLICATIONS RECEIVED

The Blakiston Company, Philadelphia: How to Become a Doctor, by George R. Moon, A. B., M. A.

Grune & Stratton, Inc., New York: Cardiovascular Disease, by Louis H. Sigler, M. D., F. A. C. P.

Paul B. Hoeber, Inc., New York: Hematology, by Willis M. Fowler, M. D. (Revised Second Edition).

Oxford University Press, New York: Dutch Archives of Surgery.

W. B. Saunders, Philadelphia: Oral and Dental Diagnosis, Third Edition, by Kurk H. Thomas, D. M. D., F. D. S. R. C. S. Eng. Geriatric Medicine, Second Edition, edited by Edward J. Stieglitz, M. S., M. D., F. A. C. P. Psychosomatic Medicine, 2nd Edition, by Edward Weiss, M. D. and O. Spurgeon English, M. D. Medical Etymology, by O. H. Perry Pepper, M. D.

Charles C. Thomas, Springfield, Ill.: Obesity, by Edward H. Rynearson, M. D., F. A. C. P., and Clifford F. Gastineau, M. D.